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THE USE OF RADIOACTIVE CHROMIUM (^{51}Cr) IN CLINICAL MEDICINE.

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THE labelling of red blood cells with ^{51}Cr was introduced by Gray and Sterling (1950*a*), who used the labelled cells for the estimation of the volume of the circulating red cells. Subsequently, ^{51}Cr labelled cells were used by Ebaugh, Emerson and Ross (1953) for the determination of red cell life span.

The ^{51}Cr technique for the determination of blood volume and red cell survival has been used in the Hæmatology Clinical Research Unit at St. Vincent's Hospital since 1955. It is the purpose of this paper to describe the technique used, and to indicate the practical application of these determinations, with particular reference to the importance of blood volume determinations in the differential diagnosis of *polycythæmia vera* and polycythæmia of stress.

METHOD.

The method described here is based on that of Mollison and Veall (1955), whose paper should be consulted for a more detailed discussion.

Normal red cells contain 20 microgrammes of chromium per 100 millilitres of packed red cells (Gray and Sterling, 1950*b*). When chromium is added to blood as sodium chromate in the hexavalent form, it is rapidly taken up into the red cells. It is suggested that in the red cell the sodium chromate is converted to the trivalent form, and that it rapidly combines with the globin fraction of hæmoglobin. In this form it cannot pass freely out through the red cell membrane, and it is not reutilized when the red cells are destroyed. Red cells can take up about 400 times their normal content of chromium without any measurable effect on their viability.

For these reasons, the radioisotope ^{51}Cr is a most satisfactory isotope for labelling red cells in the estimation of blood volume and of red cell life span. The isotope is readily taken up by the red cell, in which it is retained during the life of the cell, only a small percentage being lost by elution. The rate of elution appears to be constant, and so the true survival curve of the red cells can be calculated.

Blood Sample.

The method of dealing with the blood sample is as follows.

Ten to 14 millilitres of blood are taken from the patient with a dry sterile syringe and introduced into a sterile one-ounce universal container (McCartney bottle) containing three millilitres of acid-citrate-dextrose (A.C.D.) solution. A.C.D. is preferred to heparin as an anticoagulant, because there is a greater uptake of the radioactive chromium into the red cells in a slightly acid solution (Necheles, Weinstein and LeRoy, 1953). The blood

[1] Working with the aid of a grant from the National Health and Medical Research Council of Australia.

sample is then centrifuged at 3000 revolutions per minute for 10 minutes, after which time the supernatant "A.C.D.-plasma" is removed and kept under sterile conditions, thus leaving the packed red cells ready for labelling.

Radioactive Chromium (^{51}Cr).

In Great Britain the largest permissible dose of ^{51}Cr for an adult is 100 microcuries (Mollison, 1956). This amount is still well below the tolerance dose. In red-cell survival studies, it is advisable to use the largest dose possible, as the cells can then be followed for the maximum length of time. If 120 microcuries of ^{51}Cr are added to the packed red cells and four-fifths of these tagged cells are injected (see below), then the maximum dose of 100 microcuries is not exceeded. On the other hand, in the estimation of blood volume the red cells are not traced after the first day, and so smaller doses (50 to 70 microcuries) can be used.

The radioactive sodium chromate solution is prepared at the Atomic Energy Research Establishment, Harwell, United Kingdom, and is distributed in this country by the Commonwealth X-ray and Radium Laboratories, Melbourne. It is dispensed into an ampoule to contain the desired amount of radioactivity for the day required and then sterilized in an autoclave. The ampoule as dispatched is sterile.

Labelling the Red Cells.

The dose of ^{51}Cr for red-cell survival studies is 120 microcuries, and for blood volume estimation is 70 microcuries. The dose of ^{51}Cr is transferred with a sterile Pasteur pipette from the ampoule into the vessel containing the packed red cells to be labelled. There is a significantly greater uptake of ^{51}Cr when it is added to the packed cells than when it is added to the whole blood (Mollison and Veall, 1955). After thorough mixing, the vessel is allowed to stand at room temperature for 30 minutes. With the abovementioned procedure, the uptake of radioactive chromium by the red cells ranges from 90% to 95%.

The specific activity of the ^{51}Cr used in estimations done in this laboratory has ranged from 1·6 microcuries per microgramme to 19 microcuries per microgramme. The maximum amount of chromium added to the packed cells was seven microgrammes per millilitre of packed cells. It has been shown that doses of between 0·6 microgramme and 20 microgrammes of chromium per millilitre of red cells do not damage normal red cells (Hughes, Jones and Mollison, 1956; Donohue *et alii*, 1955).

Washing of Labelled Cells.

After packed cells have stood for half an hour, they are washed once or twice in a 2% solution of A.C.D. plasma-saline mixture, prepared by adding two millilitres of the supernatant plasma previously removed from the red cells to 100 millilitres of 0·85% saline for intravenous use.

After one wash (that is, filling the vessel with plasma-saline, mixing thoroughly, centrifuging, discarding the supernatant and again filling the vessel with plasma-saline ready for injection), the supernatant activity of the injection suspension (see below) ranged from 1·1% to 1·8% of the total activity, with a mean of 1·3%. After two washes, the supernatant activity ranged from less than 0·1% to 0·5%, with a mean of 0·24%.

When estimating blood volume, it is advisable to wash the cells twice, as this removes almost all of the supernatant activity. However, in estimating the survival time of red cells from patients with severe hæmolytic anæmia, we believe that one wash is advisable, as it lessens the in-vitro handling of the fragile red cells.

Injection of Tagged Cells.

After these cells have been washed, the injection suspension is prepared by making up the packed red cells to a volume of 25 millilitres with the addition of plasma-saline and mixing them thoroughly together. A 20 millilitres calibrated syringe is filled exactly to the 20 millilitres mark with this suspension. Approximately two millilitres of the injection suspension are kept for preparation of the standard, and the remainder is centrifuged, the supernatant being removed and kept for estimation of extracellular activity.

A 20 gauge needle attached to a five millilitres syringe is inserted into the vein of the patient. When it is assured that the needle is in the vein, the five millilitres syringe is detached, the 20 millilitres syringe containing the labelled cells is attached and the contents are injected.

Sampling.

Ten minutes after the injection, 20 millilitres of blood are taken from the patient with a syringe wet with herapin.[1] Duplicate hæmatocrit estimations are calculated for the sample. The hæmatocrit tubes are centrifuged for 55 minutes at 3000 revolutions per minute in a centrifuge of radius 15 centimetres; the plasma trapped in the red-cell column can then be calculated (Chaplin and Mollison, 1952), and the true hæmatocrit reading of the blood can be determined. When the survival of the red cells is being followed over a period of time, the result of the radioactive counts of the sample on any day is corrected for the change in hæmatocrit estimation between that sample and the ten-minute sample (see calculation). The application of this correction for change of hæmatocrit reading assumes that any small changes in it are due to changes in the plasma volume. However, if this variation is due to changes in the patient's red-cell volume, as a result of variation either in the rate of production or more particularly in the rate of destruction, then the correction for change in hæmatocrit reading no longer applies.

For total red-cell volume estimations, one blood sample taken from the patient 10 minutes after injection is all that is required. For red-cell survival estimation a sample should be taken 10 minutes, 24 hours and 48 hours after injection, and thereafter as considered necessary, according to the rate of disappearance of the cells. The activity in the sample taken 10 minutes after injection is taken as 100% survival.

Preparation of Samples for Counting.

After the removal of an aliquot of blood for hæmatocrit estimations, the remainder of the sample is hæmolysed with saponin (about as much as will fit on the end of a spatula). This blood sample is now ready for counting without any dilution.

The standard is prepared by the dilution of the injection suspension in the ratio of one in 200 in distilled water (0·5 millilitre into a 100 millilitres standard flask). The supernatant of the injection solution is diluted one in 20 in distilled water (that is, one millilitre into a 20 millilitres standard flask). A solidified ^{51}Cr standard is prepared and used as described by Hughes Jones and Mollison (1956).

Counting.

The radioactivity counts are made on a gamma ray scintillation counter using a thallium-activated sodium iodide crystal. The instrument used in this department is a Burndept directional scintillation counter modified to take an annular cup for liquid counting. The plastic cup was made by C. Alger and Sons, Melbourne; it holds about 17 millilitres of blood. A pipette calibrated to contain 17·6 millilitres is set up with a three-way tap attached to a suction pump, so that the blood can be drawn up, held at the mark and then delivered automatically. The pipette can be cleaned and dried *in situ* by running through it clean water and then acetone with the tap turned to the suction pump.

For the total red-cell volume estimations, the blood standard is counted before and after the ten-minute blood sample is counted. This is an assurance against variation in the machine while the samples are being counted. The two counts are averaged, and the mean is taken as the standard count.

For the estimation of the red cell survival time, the solidified ^{51}Cr standard is counted each day, before and after the blood sample is counted. By referring to this standard, the counts of the blood sample can be corrected for radioactive decay and for variation in the machine, and so the fall in radioactivity due to loss of red cells can be calculated.

The background activity is counted before and after each set of counts. This background is subtracted from the counts before calculations are made.

Calculation.

Blood Volume.

The total red cell volume can be calculated from equation "A".

This will give the total red cell volume in millilitres. To express the red cell volume in millilitres per kilogram of body weight, the total red cell volume is divided by the patient's weight in kilograms.

[1] An amount of 0·08 millilitre (two to three drops) of heparin solution containing 25,000 immunizing units per millilitre is more than sufficient to prevent 20 millilitres of blood from clotting.

"A"

$$\text{Total red cell volume} = \frac{\text{Volume injected (millilitres)} \times \text{Radioactive counts per minute of standard} \times \text{Dilution of standard} \times \text{Hæmatocrit corrected for trapped plasma (as a fraction of 1)} \times \left(1 - \frac{\%SN}{100}\right)}{\text{Radioactive counts per minute of blood sample}}$$

$$\%SN = \frac{\text{Counts per minute of supernatant}}{\text{Counts per minute of standard}} \times \frac{\text{Dilution of supernatant}}{\text{Dilution of standard}} \times 100$$

The total blood volume is calculated from the total red cell volume as follows:

$$\text{Total blood volume} = \frac{\text{Total red cell volume}}{\text{Venous hæmatocrit corrected for trapped plasma} \times 0{\cdot}91} \times 100.$$

Venous hæmatocrit (corrected) $\times 0{\cdot}91$ = body hæmatocrit (see Mollison, 1956, page 63).

The plasma volume can be calculated by subtracting the total red cell volume from the total blood volume.

Example.—The following is an example of a calculation of the blood volume.

Volume injected	20·0 millilitres
Radioactive counts per minute of standard	3324
Dilution of standard	200
Hæmatocrit corrected for trapped plasma	45·5%
Hæmatocrit corrected for trapped plasma as a fraction of 1	0·455
Radioactive count per minute of blood sample	3400
Counts per minute of supernatant	64
Dilution of supernatant	20
Weight of patient	62·0 kilograms

Red cell volume

$$= \frac{20{\cdot}0 \text{ millilitres} \times 3324 \times 200 \times 0{\cdot}455 \times \left(1 - \frac{\%SN}{100}\right)}{3400}$$

$$\text{Percentage } SN = \frac{64 \times 20}{3324 \times 200 \times 100} = 0{\cdot}19\%$$

$$= \frac{20{\cdot}0 \times 3324 \times 200 \times 0{\cdot}455 \times 0{\cdot}998}{3400}$$

$$= 1770 \text{ millilitres}$$

$$= \frac{1770}{62} \text{ millilitres per kilogram}$$

$$= 28{\cdot}5 \text{ millilitres per kilogram}$$

Total blood volume

$$= \frac{1770}{45{\cdot}5 \times 0{\cdot}91} \times 100$$

$$= 4270 \text{ millilitres}$$

$$= 68{\cdot}9 \text{ millilitres per kilogram}$$

Plasma volume

$$= 2500 \text{ millilitres}$$

$$= 40{\cdot}4 \text{ millilitres per kilogram}$$

Normal Values.—Some of the published values for mean normal total red cell volume, plasma volume and total blood volume are given in Table I.

The range of normal values for the red-cell volume for adult males in our laboratory is 26·3 to 32·3 millilitres per kilogram, and that given by Berlin *et alii* (1950) is 22·8 to 35·8 millilitres per kilogram. Huff and Feller (1956) state that the values for 95% of the population lie between 20 millilitres and 38 millilitres per kilogram for men and 19 millilitres and 29 millilitres per kilogram for women, and that the diagnosis of an increase in circulating red cell volume requires a value greater than 38 millilitres per kilogram in men and 29 millilitres per kilogram in women.

The range of normal values for the plasma volume for adult males in our laboratory is 36·5 to 49·5 millilitres per kilogram, and that given by Berlin *et alii* (1950) is 32·5 to 45·1 millilitres per kilogram. The values for normal adult females given by Berlin *et alii* (1951) range from 27·3 to 46·6 millilitres per kilogram.

TABLE I.

Mean Blood Volumes in Normal Male and Female Subjects.

Subjects.	Authors.	Total Red Cell Volume. (Millilitres per Kilogram.)	Plasma Volume. (Millilitres per Kilogram.)	Total Blood Volume. (Millilitres per Kilogram.)
Adult males.	Berlin *et alii* (1950)	29·9	38·7	—
	Reeve and Veall (1949)	30·0	46·6	76·6
	Giblett *et alii* (1956)	26·5	—	66·0
	Huff and Feller (1956)	28·27	33·45	61·54
	Present authors	27·7	41·3	69·4
Adult females.	Berlin *et alii* (1951)	27·0	37·0	64·4
	Wadsworth (1954)	23·4	43·1	66·5
	Huff and Feller (1956)	24·24	34·8	58·9

Red-Cell Survival.

The count of the ten-minute sample (day 0) is taken as 100% red cell survival. Counts on subsequent days (e.g. day y) are estimated as a percentage of this value; this value is referred to as "percentage ^{51}Cr survival" (equation "B").

This calculation applies if the blood sample is taken and counted on day y. If the blood sample is taken on day y and counted on day z, then the extra radioactive decay must be allowed for, and the equation reads as in "C".

The values for percentage ^{51}Cr survival can be plotted against time (in days) on arithmetic paper. The time at which the curve crosses the 50% ^{51}Cr survival is called the 50% ^{51}Cr survival time.

Hughes Jones and Mollison (1956) have given an equation for the estimation of true red-cell survival (see equation "D").

"B"

$$\text{Percentage } ^{51}\text{Cr survival on day } y = \frac{\text{Radioactive counts per minute of sample day } y}{\text{Radioactive counts per minute of sample day 0}} \times \frac{\text{Expected counts of solidified } ^{51}\text{Cr standard day } y}{\text{Actual counts of solidified } ^{51}\text{Cr standard day } y} \times \frac{\text{Corrected hæmatocrit day 0}}{\text{Corrected hæmatocrit day } y} \times \frac{100}{\text{Percentage radioactive decay on day } y} \times 100$$

"C"

$$\text{Percentage } ^{51}\text{Cr survival on day } y = \frac{\text{Radioactive counts per minute of sample day } y}{\text{Radioactive counts per minute of sample day 0}} \times \frac{\text{Expected counts of solidified } ^{51}\text{Cr standard day } z}{\text{Actual counts solidified } ^{51}\text{Cr standard day } z} \times \frac{\text{Corrected hæmatocrit day 0}}{\text{Corrected hæmatocrit day } y} \times \frac{100}{\text{Percentage radioactive decay on day } z} \times 100$$

"D"

$$\text{Percentage red cell survival on day } y = \frac{\text{Observed } ^{51}\text{Cr survival (\%) on day } y}{\text{Normal } ^{51}\text{Cr survival (\%) on day } y} \times \frac{110 - y}{110} \times 100.$$

These values so obtained can be plotted on arithmetic paper against time (in days), and then the true red-cell survival curves can be obtained. The normal red-cell survival curve is a straight line which cuts the time axis at 110 days.

Example.—In the following example, all counts have had the background subtracted.

10-minute sample (100%):

Counted on day of injection (day 0) .. .. .. = 7359 counts per minute
Corrected hæmatocrit = 42·8%
^{51}Cr standard count .. .. = 13,676 counts per minute

8-day sample:

Count on 8th day (day y) = 1998 counts per minute
Corrected hæmatocrit .. = 41·1%
Actual ^{51}Cr standard count = 11,258 counts per minute

$$\text{Expected } ^{51}\text{Cr standard count} = 13{,}676 \times \frac{\text{decay of } ^{51}\text{Cr in 8 days}}{100}$$

$$= 11{,}201 \text{ counts per minute}$$

$$\text{Percentage } ^{51}\text{Cr survival of red cells on 8th day} = \frac{1998}{7359} \times \frac{11{,}201}{11{,}258} \times \frac{42{\cdot}8}{41{\cdot}1} \times \frac{100}{81{\cdot}9} \times 100$$

$$= 34{\cdot}4\%.$$

If the above sample is collected on the 8th day (day y) but not counted until the next day (day z

Count on 9th day (day z) = 1948
Corrected hæmatocrit .. = 41·1%
Actual ^{51}Cr standard count = 10,824

$$\text{Expected } ^{51}\text{Cr standard count} = 13{,}676 \times \frac{\text{decay of } ^{51}\text{Cr in 9 days}}{100}$$

$$= 10{,}927$$

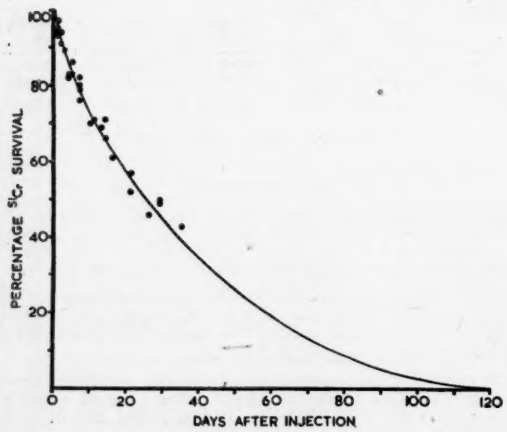

FIGURE I.

This figure illustrates the rate of disappearance of normal cells labelled with ^{51}Cr. The line represents the values given by Mollison and Veall (1955), and the points are the values obtained in the present study.

$$\text{Percentage } ^{51}\text{Cr survival of red cells on 8th day} = \frac{1948}{7359} \times \frac{10{,}927}{10{,}824} \times \frac{42{\cdot}8}{41{\cdot}1} \times \frac{100}{79{\cdot}9} \times 100$$

$$= 34{\cdot}7$$

$$\text{Percentage survival of red cells on 8th day} = \frac{34{\cdot}7}{76} \times \frac{(110-8)}{110} \times 100$$

$$= 42\%.$$

Normal Values.—Some of the published values for 50% ^{51}Cr survival time are as follows: Read *et alii* (1954), 26 days; Mollison and Veall (1955), 25 days ± 5·5%; Donohue *et alii* (1955), 26 days ± 2 days; present authors, 26 days ± 3 days.

The ^{51}Cr survival curve shown in Figure I is taken from Mollison and Veall (1955), and the points on the figure are the normal values obtained in the present work.

The survival curves of two patients are shown in Figures II and III. Figure II gives the ^{51}Cr survival curves, Case I giving a 50% ^{51}Cr survival time of 21 days, and Case II of 14 days. Figure III shows these same results converted to true cell life. If the line joining the points is extrapolated to the time axis, then the mean cell life for Case I is 77 days and for Case II 40 days.

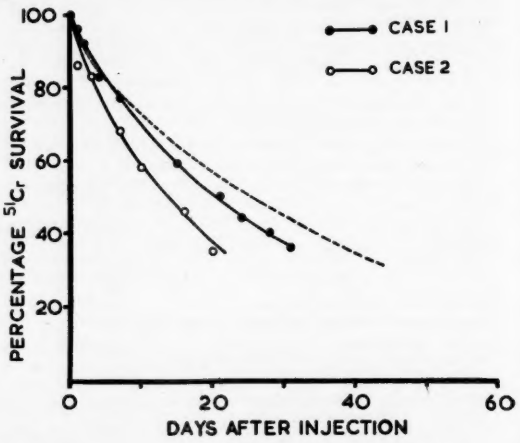

FIGURE II.

This figure illustrates the ^{51}Cr survival curves of two subjects with hereditary elliptocytosis. In Case I, although there is no clinical evidence of hæmolytic anæmia, the 50% ^{51}Cr survival time is shortened to 21 days. In Case II there is frank clinical and hæmatological evidence of hæmolytic anæmia, and the 50% ^{51}Cr survival time is 14 days. The interrupted line gives the ^{51}Cr survival curve of normal cells.

DISCUSSION.

Blood Volume Determinations in the Diagnosis of Polycythæmia.

Blood volume determinations have proved to be of great value in the diagnosis of polycythæmia, particularly in the differentiation of *polycythæmia vera* and polycythæmia of stress. When *polycythæmia vera* is in its early stages or is "masked" by an increased plasma volume, the red-cell values of the peripheral blood may be only slightly increased or may even be in the upper normal range. Thus diagnosis from polycythæmia of stress may be difficult, especially as the florid complexion in the latter condition may be indistinguishable from that of *polycythæmia vera*. Accurate diagnosis of these disorders is of especial importance because of the difference in treatment and prognosis.

Polycythæmia of Stress.

Polycythæmia of stress has only recently been characterized, and it will therefore be discussed in some detail. It is a relative polycythæmia of unknown ætiology, and was first described in 1952 by Lawrence and Berlin. They called it "polycythæmia of stress", as about half their patients had an anxiety state or were mildly psychoneurotic, and it was thought that the condition might be related to nervous stress. It has also been referred to as "stress erythrocytosis" (Dameshek, 1953), but this term is not strictly accurate, as, by definition, erythrocytosis is a true but not a relative polycythæmia. Polycythæmia of stress occurs much more commonly in males than in females, and although it may occur at any age in adult life, it is most frequently seen in middle-aged persons. There is no typical history. Symptoms of an anxiety state, e.g. fatigue, irritability, headache and nervousness, are common, and some patients complain of dizziness. Hypertension or obesity is present in about 50% of cases. The facial complexion is florid, often being indistinguishable from that of *polycythæmia vera*, and dilatation of the superficial vessels about the cheeks and nose is frequently present. The liver and spleen are not palpable. Red-cell values are

TABLE II.
Polycythæmia of Stress.

Case Number.	Clinical Features.	Hæmoglobin Value. (Grammes per 100 Millilitres.)	Packed Cell Volume. (Percentage.)	Blood Volume. (Millilitres per Kilogram.)	Red-Cell Volume. (Millilitres per Kilogram.)	Plasma Volume. (Millilitres per Kilogram.)
I	Male, aged 45 years. Heavy alcohol intake. Complexion florid. Spleen not palpable. White cell and platelet count normal. Weight 164 pounds	20·0	60·0	64·8	34·2	30·6
II	Male, aged 62 years. Tiredness, nervousness and irritability. Complexion florid. Spleen not palpable. White cell and platelet count normal. Weight, 204 pounds	18·4	59·0	60·3	31·2	29·1
III	Male, aged 46 years. Complexion florid. Spleen not palpable. White cell and platelet counts normal. Weight, 228 pounds	18·0	52·0	58·4	27·3	31·1
Normal values (Berlin *et alii*, 1950)	.. ..	13·5–18·0	40·0–54·0	—	Mean: 29·9 Range: 22·8–35·8	Mean: 38·7 Range: 32·5–45·1

usually at about the upper limit of normal or are slightly increased. Thus red-cell counts of 6,500,000 to 7,000,000 per cubic millimetre, hæmoglobin values of 18 to 20 grammes per 100 millilitres and hæmatocrit values of 54% to 60% are the rule, although both lower and higher values may occur. The white-cell and platelet counts are normal. The total red-cell volume is normal, the increase in concentration of the red cells being due to a pathologically low plasma volume, the cause of which is unknown. The arterial oxygen saturation is normal.

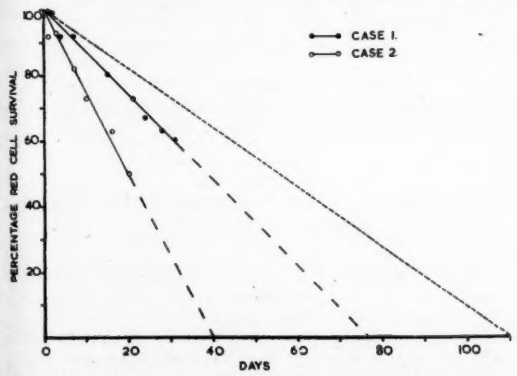

FIGURE III.

This figure illustrates the true red cell survival curves calculated from the ^{51}Cr survival curves shown in Figure II. The calculated mean cell life for Case I is 77 days, and for Case II 40 days. The interrupted line represents the survival curve of normal red cells.

The bone marrow aspirate is of normal cellularity. There is no specific treatment; hypertension, obesity and an anxiety state, when present, require their own management.

It is probable that some cases of Gaisbock's syndrome (polycythæmia with hypertension and no splenomegaly) are actually examples of polycythæmia of stress.

Table II sets out the hæmatological and blood volume studies in three patients with polycythæmia of stress. Cases I and III had been diagnosed as *polycythæmia vera*, and the patients had been referred for treatment with radioactive phosphorus.

Polycythæmia Vera.

Diagnosis of fully developed *polycythæmia vera* is usually obvious from the presence of the classical triad—a florid, brick-red complexion, splenomegaly and polycythæmia with leucocytosis and thrombocytosis. However, diagnostic difficulty may occur (i) in early cases, when red cell values are not markedly raised, and (ii) when polycythæmia is "masked" by a rise in plasma volume, which causes hæmodilution and a consequent fall in red-cell values. An increased plasma volume occurs most typically when polycythæmia is complicated by congestive cardiac failure (Guntib and Paul, 1955), but may also occur without cardiac failure (Abbatt *et alii*, 1954). In some cases of early or "masked" polycythæmia, the presence of other features of *polycythæmia vera*, e.g., splenomegaly, pruritus, leucocytosis and thrombocytosis, suggests the correct diagnosis; however, as any or all of these may be absent, especially in the early stages, diagnosis can often be made with certainty only by accurate blood-volume determinations.

Table III lists the results of blood-volume determinations in eight cases of *polycythæmia vera*. In Cases I to V no treatment had been given, and the diagnosis was clear because of the clinical and hæmatological features present. The patient in Case VI was in clinical remission after ^{32}P therapy. In Cases VII and VIII the polycythæmia was masked by an increased

TABLE III.
Polycythæmia Vera.

Case Number.	Sex.	Age. (Years.)	Hæmoglobin Value. (Grammes per 100 Millilitres.)	Packed Cell Volume. (Percentage.)	Blood Volume. (Millilitres per Kilogram.)	Red-Cell Volume. (Millilitres per Kilogram.)	Plasma Volume. (Millilitres per Kilogram.)
I	M.	60	23·7	75·0	113·0	78·0	35·0
II	M.	47	24·0	74·0	120·0	78·0	42·0
III	F.	68	18·5	64·0	104·3	59·5	44·8
IV	M.	63	20·5	70·0	103·6	70·5	33·1
V	M.	57	19·9	76·0	114·4	79·7	34·7
VI	M.	62	17·4	57·0	74·1	38·5	35·6
VII	F.	60	15·2	47·0	100·0	42·1	57·9
VIII	M.	60	11·7	45·0	129·9	52·8	77·1

plasma volume. The patient in Case VII was an untreated patient with peripheral blood red-cell values at the upper limit of normal; blood-volume studies showed that her red-cell volume was significantly increased, and they established the diagnosis of true polycythæmia. The patient in Case VIII, in remission after ^{32}P therapy, developed congestive cardiac failure; examination of the peripheral blood revealed an iron deficiency anæmia, but blood-volume studies showed a significant increase in red-cell volume and a marked increase in plasma volume.

Red-Cell Survival Estimations.

The determination of red-cell life span is more important as a research tool in the investigation of hæmolytic anæmia than as a routine method in clinical medicine. Nevertheless, it is being increasingly used in clinical work (*a*) to determine whether an anæmia of uncertain ætiology is hæmolytic in type, and (*b*) to assess the hæmolytic element, and thus the possible response to splenectomy, in certain anæmias in which both impairment of production and hæmolysis contribute to the anæmia, e.g., myelosclerosis.

Until the introduction of ^{51}Cr, the Ashby technique of differential agglutination was the most satisfactory method for determining red-cell life span. This method has certain disadvantages: it requires a blood transfusion with the attendant risk of homologous serum jaundice; large volumes of donor blood (approximately 500 millilitres) must be used; the survival of the patient's cells cannot be followed in his own circulation; and its use is limited by the availability of suitable antisera. On the other hand, the ^{51}Cr method does not require transfusion, only small volumes of blood are necessary, survival of the patient's cells can be followed in his own circulation and it can be used in patients of all blood groups. It is of particular value in the estimation of red-cell life span in patients with little or no anæmia who, therefore, do not require transfusion. For the abovementioned reasons, the ^{51}Cr method is now the method of choice for determination of red-cell life span in clinical practice.

Other Uses of ^{51}Cr.

In addition to its main use for the determination of blood volume and red-cell survival, the labelling of red cells with ^{51}Cr has recently found other applications. Jandl *et alii* (1956) used this method to determine the in-vivo sites of red-cell destruction, and found it of use in the prediction of the probable response to splenectomy in certain hæmolytic anæmias. Cooper *et alii* (1955) used it as a method for the quantitative determination of bleeding into the alimentary tract. ^{51}Cr has also been used for the labelling of leucocytes by McCall *et alii* (1955), and for the labelling of platelets by Morgan *et alii* (1955).

Summary.

The radioactive chromium technique for the estimation of blood volume and red-cell life span is described. The application of this technique in clinical medicine is discussed, with particular reference to the use of blood volume determinations in the differentiation between polycythæmia of stress and *polycythæmia vera*.

Acknowledgements.

We are very much indebted to Mr. D. J. Stevens and Miss V. Sheriff, of the Commonwealth X-ray and Radium Laboratory, for their advice and practical assistance.

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Addendum.

It is probable that the use of a well-type scintillation crystal will enable smaller doses of ^{51}Cr to be used.

NEW SURGICAL TREATMENTS OF DEAFNESS SIMPLY EXPLAINED.

By A. B. K. WATKINS, M.S. (London), F.R.C.S. (England), *Sydney*.

HALF a decade has passed since I wrote an article for the purpose of explaining the fenestration operation simply for practitioners not engaged in ear, nose and throat practice. At that time not much surgery, apart from the fenestration operation, was being performed for the avowed purpose of curing deafness, though naturally the surgical cure of suppurative conditions in the ear did, incidentally, often produce improved hearing.

Of late years, considerable effort and research have been directed towards the surgical relief of deafness, and progress has been so great that it can almost be compared with the progress of atomic physics. Not only have other operations now been added to that of fenestratio.. for the treatment of otosclerosis, but a number of other operations are in routine use for the cure of other forms of deafness.

In my previous communication I explained the mechanism of deafness in otosclerosis, and that the disease consists of a spongification of the bone of the capsule of the internal ear; this does not produce deafness until the disease spreads and causes fixation of the foot plate of the stapes in the oval window, or causes closure of the round window. As the disease progresses, an associated cochlear degeneration often develops. I also explained how fixation of the stapes (and certain rarer conditions) prevents airborne sounds from reaching the inner ear, whilst it allows bone-conducted sounds to pass almost normally. This results when a 256 vibrations per second tuning fork is applied to the mastoid; vibrations are heard much longer than when the tuning fork is held opposite the meatus. It also often produces the condition known as "*paracusis Willisii*"; the patient hears conversation better when there is a loud background noise (e.g. in a factory or in a noisy vehicle). This symptom is not to be expected in the very early stage of otosclerosis, when only one ear is involved,

whilst the patient is wearing a hearing aid or when the disease has advanced to the stage of extreme cochlear degeneration.

I pointed out that most patients suitable for fenestration exhibited paracusis. I advised that all patients with that symptom should be encouraged to obtain an expert opinion as to whether they might benefit from fenestration. Because of subsequent advances in otology, I would now extend this former advice to include, besides patients with paracusis, those with chronic subtotal perforations of the tympanic membrane (especially if dry), and those patients who can hear the sound of a 256 vibrations per second tuning fork very much longer on the mastoid than opposite the meatus.

Tympanic Membrane Perforations.

A chronic perforation may lead to recurrent attacks of suppurative *otitis media*, either by infection through the perforation or through the Eustachian tube, up which considerable draughts of air may be expelled during nose-blowing in acute nasal infection. In fact, an occasional infection at the very least is almost inevitable. When the perforation is "central", that is, when no portion of the perforation reaches the margin of the drum, it can frequently be induced to close by a course of chemical cautery, which, of course, must be applied in concentrations and frequencies which will not cause the perforation to enlarge. By this treatment even large perforations can be induced to heal. Other cases are better treated with a simple skin graft after the edges of the perforation have been made raw by manipulation under microscope supervision. When healing of the membrane occurs after such treatment, not only is there usually a considerable improvement in the patient's hearing, but he can resume a normal existence, including swimming, without fearing (as formerly) that the ear will become reinfected.

Tympanoplasty.

In some of these patients, the nature of deafness suggests that there may be a break in the chain of ossicles, or that perhaps the chain may be fixed or that the stapes may be fixed in the oval window. In such cases, repair of the tympanic membrane alone cannot greatly improve the hearing because sound conduction by air will often still be defective. Fortunately, these lesions can now be diagnosed with the aid of a modern invention called the "audiometric probe". This consists of a wire at one end of which is a small, rounded bulb, which can be applied to the meatal wall, to the malleus or, in some cases with large perforations, to the stapes. The other end of the probe enters a device not unlike the earpiece of a hearing aid to which, in turn, is connected an audiometer, which causes it to vibrate at various pitches and at various amplitudes. By comparing loudness of the sounds produced by the probe in the various positions mentioned, the surgeon obtains exact data on what he may expect to find at operation.

If the perforation happens to be "lateral" (that is, reach the periphery of the drum), or if abnormalities of the ossicular chain are present in patients with central perforations, more extensive operative procedures than simple closure of the perforation are necessary. To do this, exploration is carried out as in a conservative radical mastoidectomy, but with a number of possible variations which are classed under the term "tympanoplasty". In some cases, little more than a skin graft closure is necessary. In others, the head of the malleus and the incus may have been removed, and the tympanic membrane is grafted directly onto the stapes (the so-called columella effect). But if the stapes is found to be fixed, those procedures advocated for otosclerosis may have to be performed. These manipulations call for the greatest manual dexterity and require the help of magnification.

Otosclerosis.

Such intense research into the fenestration operation preceded my previous article that I can honestly say that remarkably little real progress has since been made. The position is similar to that of the automobile engine which has not been greatly improved, except in detail, over the last 40 years. Nevertheless, certain points which present a nuisance value in fenestrations have perhaps become more generally recognized. For instance, the operation entails a good deal of after-treatment, and the country patient may have to arrange to be in the district where the surgeon practises for perhaps two months. The cavity in the ear has a tendency to develop recurrent superficial infections, and now and then one encounters a patient who has a permanent aural discharge or one which recurs easily. However, the bugbear of vertigo after the operation seems to have been over-emphasized. As a matter of fact, many surgeons find that a proportion of their patients deny any subsequent vertigo, whilst those who admit some vertigo seldom have enough to worry them after a week.

A simpler substitute for fenestration would be welcomed. Fortunately such an operation is being used extensively.

Stapes Mobilization (Stapediolosis).

In this operation, instead of a new oval window being made, as in the fenestration operation, the attack is made on the stapes in an attempt to make it mobile again in the old *fenestra ovalis*. The procedure, which calls for at

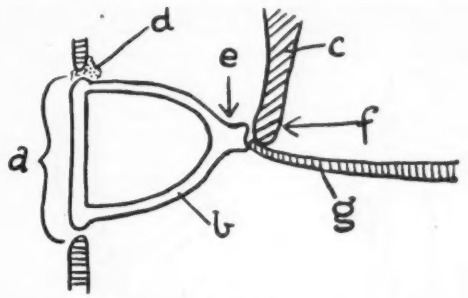

FIGURE I.

Diagram illustrating mobilization of stapes. a, foramen ovale; b, stapes; c, process of incus; d, ankylosis at anterior end of foot plate of stapes; e, place and direction of pressure in Rosen's method; f, position and direction of pressure through incudo-stapedial joint; g, position and direction of pressure through curved probe inserted into joint cavity.

least as much dexterity and as good eyesight as a fenestration, is a minor one. General anæsthesia is usually dispensed with; this is desirable in order to allow the patients' hearing to be tested during the operation. There is seldom even minor vertigo afterwards, and some patients only require to be off-duty for a long week-end to have the procedure performed. The ear heals within a few days, and subsequently there appears to be no tendency to post-operative meatitis.

Various incisions have been advocated, but the routine one at present is a slightly curved, more or less vertical incision in the posterior meatal wall a few millimetres from the tympanic membrane. This flap, with the drum attached, is folded forwards, and a very small piece of bony meatal wall has to be removed, in most cases, to allow enough of the incus and stapes to come into view.

The original method (Rosen's) was to place a tiny probe against the neck of the stapes, and to try and rock it in an antero-posterior direction. If the ankylosing bone fractures, the required result has been obtaned, but unfortunately the crura of the stapes may fracture first. More recently it has been found that greater force can safely be applied by pressing along the axis of each crus towards the foot plate with a pulsating motion. This can be done through the incudostapedial joint or with a probe actually inserted into the joint (Figure 1). Electric or mechanical vibrators are sometimes used to loosen the ankylosis. Should these procedures fail, one has by no means reached the end of the resources available. For instance, it has been found that the ankylosis is nearly always at the anterior end of the stapes, and some operators obtain functional results using a chisel at this spot. Another surgeon advises excising a piece of the anterior crus of the stapes with special scissors and fracturing the foot plate of the stapes

as far forwards as possible. Sound can then pass down the posterior crus, and can move the mobile piece of the foot plate which is posterior to the fracture (Figure II).

However, many of the best operators now try to lever the foot plate free directly. New procedures, such as fenestrating through the foot plate of the stapes, are being reported almost every week, and it is now uncertain what will become standard in a few years. At the end of the operation the drum is replaced, and a small dressing is applied.

Discussion: Fenestration Versus Stapes Mobilization.

At first it was claimed that no more than 30% of the attempts at stapes mobilization were successful. With further improvement in technique these claims have risen in one case to 90%. This is far too optimistic to be believable, but with improvement in technique some claims of about 50% sound reliable. Subsequent improvement in hearing has persisted for periods up to several years, but the operation is too recent for one to say more than that; also, there is nothing to prevent the repetition of the operation if necessary. A most important feature of the operation is that when stapes mobilization fails, fenestration can be performed in the usual manner a few months later. Apparently, the first operation does not complicate a subsequent fenestration.

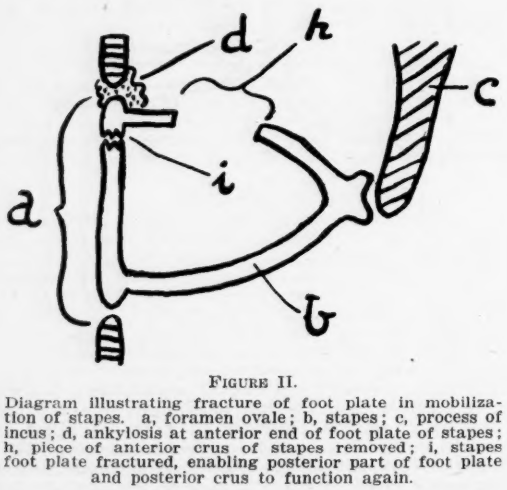

FIGURE II.

Diagram illustrating fracture of foot plate in mobilization of stapes. a, foramen ovale; b, stapes; c, process of incus; d, ankylosis at anterior end of foot plate of stapes; h, piece of anterior crus of stapes removed; i, stapes foot plate fractured, enabling posterior part of foot plate and posterior crus to function again.

Patients who obtain a satisfactory result after fenestration, though they are no longer regarded as deaf, do not regain all their hearing, especially for high notes; but after successful stapes mobilization the hearing is more nearly physiologically perfect, and the hearing of high notes is better. Further, after fenestration, patients no longer have the full normal ability to protect themselves against excessive noise; but in stapes mobilization there is no interference with this mechanism.

When cochlear degeneration is present, fenestration results are frequently disappointing, so the wary surgeon does not operate on those cases, and the extremely deaf have to be refused treatment. However, it has been shown that, after stapes mobilization, provided the mobilization is successful, some such patients obtain satisfactory hearing. There have been patients with too much cochlear degeneration to use a hearing aid who have, after operation, been able to use one again efficiently. The present tendency is to advise patients, who would previously have been recommended to submit to fenestration to have stapes mobilization attempted. This operation could also be performed on patients who would not have been advised to have fenestration, such as otosclerotics who are not yet deaf enough, and some otosclerotics with too much cochlear degeneration etc. In event of failure, fenestration can still be performed later, on all those cases considered suitable for it.

Summary.

The great progress of the surgical treatment of deafness is emphasized.

Various procedures available are referred to in a manner intended for those not practising otology.

All those patients with subtotal chronic tympanic perforations, those with symptoms of paracusis and those with much prolonged bone conduction have a considerable chance that their hearing may be improved by surgery.

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THE UBIQUITOUS STAPHYLOCOCCUS.[1]

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IT is proposed to discuss certain features of staphylococcal infection as seen in this hospital. We must stress from the start that staphylococcal infection and staphylococcal disease are not necessarily synonymous. Professor Walsh McDermott (1956) stated: "Thus, although we are gathered here to discuss something called staphylococcal infection we all know that this is not always quite the same thing as staphylococcal disease."

The problem in this hospital is probably no worse than that in most other large hospitals in Australia and elsewhere. We shall deal more particularly with the problem in the maternity section and touch but briefly on the general problem. We do not offer anything new or any answer. The maternity block is a modern, three-story hospital with 92 beds. It was completed three years ago and has approximately 2500 deliveries a year. About 75% of these are private patients.

Twelve months ago it became increasingly obvious that the incidence of staphylococcal disease as evidenced by pustules, infected umbilicuses, "sticky" eyes and paronychiæ was alarmingly high in the babies. In an attempt to elucidate the problem, the pathology department was asked to carry out a controlled survey of the situation.

In all cases, when we speak of staphylococci we refer to *Staphylococcus aureus* (coagulase-positive). All swabs taken were plated on blood agar and were then put into nutrient broth and incubated. If *S. aureus* was not grown on the original plate the broth was subcultured on to blood agar. All staphylococci isolated were tested for coagulase reaction by the slide method using human plasma. Sensitivity tests were performed against penicillin, streptomycin, chloramphenicol, oxytetracycline hydrochloride, chlortetracycline, tetracycline hydrochloride and erythromycin on all coagulase-positive *S. aureus* isolated.

RESULTS OF INITIAL SURVEY.

This survey was held over a four-week period between October 22 and November 16, 1956.

Babies.

Seventy-three babies were in hospital at the commencement of the period, and 194 babies were born during the survey. Swabs were taken from all babies with clinical signs of infection. A penicillin-resistant *S. aureus* (coagulase-positive) was grown from the swabs of 97 babies; six of these had more than one infected site. Table I shows the site and source of the "positive" swabs.

Two interesting observations emerge from Table I. (*a*) The nursing staff on the ground floor and first floor did not attempt to disinfect baths between bathing babies, and the numbers of infected umbilicuses on these two floors were far greater than on the remaining two floors, where the nurses did disinfect the

[1] Read at a meeting of the Victorian Branch of the British Medical Association, on October 12, 1957, at Geelong.

baths. (*b*) The nurses on the third floor were the only nurses who did not attempt to carry out an eye toilet, and there were far fewer infected eyes on this floor.

Nursing Staff.

The nursing staff includes trained staff, nursing aids and mothercraft nurses. All nurses available on one day had swabs taken from their noses, hands and throats; 210 swabs were taken from 70 nursing staff members. A penicillin-resistant *S. aureus* (coagulase-positive) was grown from 76 of the 210 swabs taken. The sites of "positive" swabs were the nose (45 swabs), the hands (28 swabs) and the throat (three swabs). These "positive" swabs were from 57 separate persons. In other words, 80% of the nursing staff were potential pathogen carriers. What is even more significant is that 40% of the nursing staff carried potential pathogens on their hands.

TABLE I.

Site and Source of S. Aureus Infection in Babies in Initial Survey.

Site of Infection.	Ground Floor.	First Floor.	Second Floor.	Third Floor.	Total.
Eyes	9	10	10	4	33
Umbilicus ..	17	9	2	3	31
Pustules ..	9	7	2	7	25
Nose	2	8	—	—	10
Paronychiæ ..	3	—	1	—	4

Mothers.

On one day, swabs were taken from the nose of every mother in hospital. A penicillin-resistant *S. aureus* (coagulase-positive) was grown from the swabs of 33 of the 70 mothers. In the main, classification by differential sensitivities to antibiotics of the staphylococci isolated from the mother seldom appeared to bear any relationship to that grown from her own infected baby.

Miscellaneous Areas.

Numerous areas yielded positive swabs, but the following were the most significant: "clean" babies changing benches (the nursing staff's criterion of "clean" or "dirty" was the presence or otherwise of obvious clinical infection of the babies); "clean" babies' baths; "clean" nurses' hands; sides and pans of scales; various door handles leading into nurseries; levers on wash basins; hand towels; towel rails; oil for "clean" babies; methylated spirit bottle; lid of cord tie bottle; baby suckers; cotton-wool bin; various ledges and floors; laundry tins; cot mattresses; sheets and blankets from mothers' beds; doors and cisterns of lavatories.

Differential Sensitivities of S. Aureus Isolated.

In all cases, the organism isolated was tested for sensitivity against penicillin, streptomycin, chloramphenicol, chlortetracycline, oxytetracycline hydrochloride, tetracycline hydrochloride and erythromycin. An attempt to differentiate the staphylococci into various groups by this means was not fruitful. Some information did appear to come from grouping the staphylococci into those insensitive to penicillin only and those insensitive to penicillin and other antibiotics (Table II).

TABLE II.

Grouping of S. Aureus According to Their Resistance.

Subject.	Staphylococci Resistant to Penicillin Only.	Staphylococci Resistant to Penicillin and Other Antibiotics.
Babies	54	45
Staff	30	35
Mothers	24	10
Miscellaneous areas ..	27	24
Total	135	114

Table II shows that the general pattern of resistance of the organisms isolated from the babies, staff and miscellaneous areas roughly corresponds, whereas that of the mothers is different. This tends to indicate that the main source of cross-infection is not the mothers.

Discussion.

Certain facts and impressions emerged from this survey.

1. The understanding of infection and cross-infection by the nursing staff was noticeably poor.

2. Less than 5% of the staff carried the staphylococcus in their throats, and it appeared that "droplet" infection played a very minor role in contamination of the babies. Hare and Thomas (1956) stress the point that *S. aureus* occurs so infrequently in the throat that egress from the mouth need not be considered an important factor in the dissemination of this organism.

3. Transfer of infection by the hands was accentuated by two factors. The first was the positioning of wash basins. In many cases the basins were separated by doors from the nursery or area where the staff intended to work after washing their hands. In several instances, staff were seen to wash their hands and then to open doors by handles from which *S. aureus* was later grown. The second was the use of community towels. Staphylococci could invariably be isolated from these.

4. Frequent bathing and weighing unnecessarily exposed the babies to what was generally a potent source of infection. From most baths and scales the staphylococcus was grown profusely.

5. Handling of laundry required revision. In many cases, sorting of dirty laundry took place in the nurseries, and mattresses and blankets were infrequently fumigated.

6. Overcrowding did not appear to play an important part. Overcrowding and "lack of competent staff" were often confused.

General Recommendations.

The following general recommendations were made.

1. Regular and good lectures and demonstrations are necessary for all nursing staff. The ideal measuring stick for both nursing and medical staff is that the hospital be a midwifery training school.

2. Clinical staphylococcal infection of a member of the staff must mean exclusion from the maternity wing until he or she is clear of the infection.

3. The hand-washing facilities should be improved. Positioning of wash basins needed reviewing. Hexachlorophane or similar antiseptic soap should be used for hand washing. Community towels should be replaced by paper towels or by individual disposable towels. An antiseptic hand cream such as "Hibitane" cream should be used after drying of hands. It would serve a dual purpose with its antiseptic properties and pleasantness.

4. Frequent bathing and oiling of babies should stop. In most cases the baby needs only one bath three to eight hours after birth and that should be in sterile water in a sterile bath. A demonstration bathing of the baby on the day of discharge from hospital may also be necessary in the case of mothers with first babies.

5. If toilet procedures are carried out at communal benches, this should be done on sterile paper, which is discarded after use. The paper can be newspaper.

6. Weighing of babies should be cut down to an absolute minimum. If babies are weighed, the pan should be covered with sterile paper, which is discarded after use.

7. There must be standardization in the handling of all clothing and linen whether it is obviously dirty or "clean".

8. Various floors in turn should be closed and thoroughly cleansed and aired.

9. The mothers' stay in hospital should be shortened from the standard 10 to 12 days.

10. "Rooming in" is recommended. Next to the education of the nursing staff, this is the most important recommendation, and finds itself last on the list only because it is the most difficult to implement.

The first eight recommendations were approved and introduced into the routine of the maternity wing. One floor had to be closed because of staff shortage, and this materially assisted

in carrying out recommendations. "Rooming in" was accepted in principle; acceptance in practice still awaits the purchase of suitable cots.

Results of Survey Six Months Later.

After the first survey there was an initial drop in clinical infection, but in the following months it slowly began to rise again, and six months later a similar survey showed an even worse picture. The survey was carried out in the ten-day period from April 8 to 18, 1957.

Babies.

Forty-eight babies were in hospital at the commencement of this period, and 65 babies were born during the survey. Swabs were taken from all babies showing clinical signs of infection. *S. aureus* (coagulase-positive) was grown from the swabs from 59 babies. The sources of "positive" swabs were: the eyes, 21; pustules, 21; the umbilicus, 27; paronychiæ, 9; the nose, 1. Of the staphylococci grown, 62 were penicillin-resistant. This is in slight contrast to the previous survey, in which all staphylococci were penicillin-resistant. This was not an error in the original technique, as all organisms grown then were kept, and they yielded the same results when they were retested for penicillin sensitivity with the organisms grown in this second survey.

Nursing Staff.

Swabs were taken from the hands and noses of 60 nursing staff members; 46 individuals yielded *S. aureus* (coagulase-positive). The sources of "positive" swabs were: the nose, 43; the hands, 8. Of the 51 staphylococci grown, 33 were penicillin-resistant. It was pleasing to note the marked drop in the contamination of hands amongst the nursing staff.

Table III.

Phage Types of Staphylococci Isolated from Nursing Staff.

Ground Floor.	First Floor.	Third Floor.	Delivery Ward.	Night Staff.
7	7	52A/79	52A	29
52	80/81	3A/3B/55	80/81	3B/70
3B/55	52	80/81	52/52A/80/81	29/52
80	3B/70	52/52A/80/81	7	47C/47D/81
47D	81	52A	51/52A/80/81	29/52
	52/52A/80/81	3B/55	3B/55	80/81
		52/52A/80/81	44/52A/42E/47/80	47/D

Most of the nursing staff had had swabs taken six months previously. A comparison by differential sensitivities of the staphylococci carried in the first survey showed a difference in a very high percentage of cases. This "temporary-carrier" class (persons liable to acquire and lose strains frequently) has been noted by other workers (Hutchison, Green and Grimsen, 1957). In their series only 21% persistently carried the same phage type for 17 weeks or more. Most of the staphylococci isolated from the nursing staff were phage typed.

As can be seen from Table III, the phage types were as scattered and varied as were the antibiotic sensitivities.

Mothers.

Swabs were taken from the hands and noses of 48 mothers, and of these 32 grew *S. aureus* (coagulase-positive). The sources of "positive" swabs were: the nose, 29; the hands, 6. Of the 35 staphylococci grown, only 14 were penicillin-resistant.

Miscellaneous Areas.

Fewer areas yielded positive findings compared with the previous survey. The most significant areas were: the tearing edge of the newly installed paper towel holders on two floors; the scales on two floors; some of the baths; the floors of various nurseries.

Discussion.

This second survey showed that over a period of six months the over-all picture had not improved, in fact it appeared worse. This occurred in spite of the changes and improvement in the management of this maternity block. The changes appeared to be carried out conscientiously by the nursing staff and all persons concerned.

All the staphylococci isolated were not phage typed, but it was obvious that the types were widely scattered over all floors. There was not one predominant type amongst the nursing staff. A very limited number of staphylococci isolated from infected babies were phage typed. These gave a similar scattered distribution. There did not appear to be a particular "hospital staph.". The staphylococci in the maternity wing were, on the contrary, a changing but very constant population.

The surveys were not statistically carried out, but the impression gathered is that neither the sensitivity of the organism to antibiotics nor its phage type plays very much part in its pathogenicity. We believe that, given the right conditions at the right time and in the right place, the only criterion necessary is that it be a staphylococcus. It was believed that an even bleaker picture might be obtained if swabs were taken from all babies, whether clinically infected or not.

Table IV.

Incidence of S. Aureus Grown Correlated with Age of Baby in Days.

Age in Days.	Number of Babies.	*S. aureus.*		Site.		
		Grown.	Not Grown.	Eyes.	Umbilicus.	Nose.
Less than 1	6	1	5	—	1	1
1 ..	4	2	2	—	1	1
2 ..	9	6	3	4	5	4
3 ..	12	11	1	9	9	8
4 ..	10	8	2	8	8	5
5 ..	20	20	—	24	14	18
6 ..	18	18	—	20	14	17
7 ..	17	17	—	14	14	15
8 to 14	40	38	2	38	26	35
Total ..	136	121	15	117	92	104

Complete Surveys, May and September, 1957.

On one day in May and again in September all babies in hospital had swabs taken from each eye, the nose and the umbilicus. The figures on each occasion were almost identical and have been grouped together for simplicity.

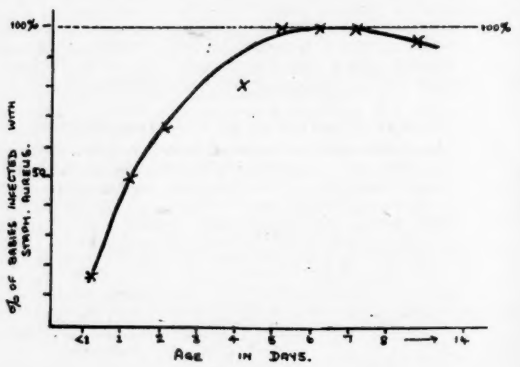

Figure I.

Altogether 544 swabs were taken from 136 babies; 121 different babies yielded *S. aureus* (coagulase-positive) from at least one site and 31 babies yielded staphylococci from all four sites. A total of 313 swabs grew *S. aureus* (coagulase-positive). Of these, some 254 staphylococci were resistant to penicillin. The sources of positive results were: the eyes, 117; the nose, 104; the umbilicus, 92. Whether the cord was still attached and its state, whether moist or dry, were recorded. These factors did not appear to bear any relationship to the organisms grown or the amount of growth. Correlation of the age of the baby with whether *S. aureus* was isolated or not yielded most interesting figures (Table IV).

These figures become even more striking when the percentage of swabs yielding *S. aureus* to the number of babies was plotted against the age in days (Figure I). The numbers are not large enough to determine whether the drop from 100% infected, which occurs in the second week, is real or accidental.

Two startling facts emerge from Table IV and Figure I. First, *S. aureus* (coagulase-positive) was grown after the second day from the eyes, nose or umbilicus in 112 out of 117 babies born, that is, 96% of the babies were cross-infected by the third day. Secondly, after the fourth day, almost 100% of the babies carried the staphylococcus in at least two and probably in three of the sites swabbed. There were 249 positive results from 95 babies.

TABLE V.

Clinical Staphylococcal Infection as Elicited by Floor Sisters.

Month.[1]	Total Number of Babies Born.	Number of Babies Swabbed.	*S. aureus* Grown.	
			Number of Babies.	Percentage of Babies Born.
January	189	45	40	21
February	190	36	32	17
March	193	43	37	19
May	179	75	53	30
June	201	69	58	29
July	179	86	75	42
August	230	85	70	30
September	207	99	78	37
Total ..	1568	538	443	28

[1] April is omitted from the figures given as one of our surveys was carried out that month.

It may be argued that the babies were born with the staphylococcus. This does happen in a very small percentage of cases, e.g. the authors grew *S. aureus* from a teaspoonful of pus removed from a "sticky" eye of a baby one hour after birth. This is the exception rather than the rule. Figure I clearly shows the increasing incidence of staphylococcal infection during the first four days of life.

TABLE VI.

Staphylococcal Disease (as Seen by Floor Sisters) Correlated with Age of Baby.

Age in Days.	Number of Babies Whose Swabs Grew *S. aureus* (Coagulase-positive).
<1	1
1	4
2	31
3	47
4	68
5	93
6	58
7	44
8 to 14	97
Total	443

Our results are representative in that there was absolutely no selection of babies, whether from private or public wards. Swabs were taken from all babies by one of us, and only one swab was taken from each site. More swabs from each site may have presented an even gloomier picture.

Staphylococcal Disease as Elicited by Floor Sisters.

After the initial survey 12 months earlier, the sister in charge of each floor was asked to take swabs from any babies when they first showed obvious signs of infection and to send these swabs for culture. The results of tests performed on these swabs are shown in Table V.

Despite continual awareness of the problem of staphylococcal infection, Table V clearly shows an increase this year of actual staphylococcal disease in the babies. The over-all figure of 28% of babies, irrespective of age, is lower than in the controlled surveys, as the foregoing figure includes babies missed by busy sisters. Also, possibly some swabs were not taken owing to pressure of work and other factors.

Correlation of the age of the baby with the number of babies showing staphylococcal disease (Table VI) presented a similar picture to that of our controlled surveys.

As will be seen, there is a startling increase in staphylococcal disease after the first day in hospital. A peak is reached at the fifth day; the decrease after the fifth day is not real, as the majority of babies had been found to be infected prior to this stage, and consequently swabs were not received from them in the older age groups.

Discussion on All Surveys.

What has been shown is nothing new. The majority of workers tackling the problem of cross-infection in general hospitals and maternity hospitals have shown it before. These surveys are presented, not because they bring forth new facts, but because the problem of the staphylococcus is something which must be kept continually before us. Its presence must not let us resignedly accept it, for its very existence is terrifying.

Take good note of Table V. These figures still existed despite rigid reforms enforced after the initial survey. It may be argued, and rightly so, that staphylococcal infection is not synonymous with staphylococcal disease and that the staphylococcus is forever ubiquitous. That is so, but so also were salmonella and shigella during the American Civil War.

Observations showed that the changes in procedure in the nursery and in the care of babies were being adhered to by both nursing and medical staff. One would have expected the changes brought in to have markedly cut down the incidence of staphylococcal infection and staphylococcal disease in the babies. At the very best an improvement occurred for only a few months. In the staphylococcus we have an atom bomb, which needs only the right conditions to set it off and cause staggering casualties.

"Rooming in" carried out properly is one partial answer. It does appear to go a long way towards cutting down the incidence of staphylococcal disease in babies.

The whole problem is much deeper than that which just relates to babies. The larger the hospitals built, the more magnificent they become and the more communal and automatic are all services and staff, the sooner will the atom bomb become a hydrogen bomb.

Perhaps, if we undertook the professional, social and economic difficulties associated with delivery of babies in private homes, they might save us, by teaching us again as a profession the first principles and lessons learnt by our fathers and lost to us in the powder of antibiotics.

Summary.

1. A review is made over a period of 12 months of the incidence of staphylococcal disease and staphylococcal infection in a maternity hospital with 92 beds.

2. *S. aureus* (coagulase-positive) was grown from 96% of babies aged more than two days.

3. During a twelve-month period, over one-third of the babies born developed clinical evidence of staphylococcal infection.

4. Despite measures introduced to combat cross-infection, the incidence of staphylococcal disease and staphylococcal infection remained high.

5. Some aspects of combating the problem are discussed.

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LUMPS IN THE BREAST.[1]

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"MEDICINE is a science of uncertainty and an art of probability." Nowhere does Sir William Osler's aphorism apply with more force than in the clinical diagnosis of lumps in the breast.

Table I provides the clinician with a list from which he may select a label for the particular lump he has found. It also gives an idea of the frequency of the various lesions, for it shows the diagnosis in 100 consecutive cases of lump in the breast operated upon at Sydney Hospital. These patients were admitted between January 1, 1956, and July 1, 1957. It will be seen that half the lumps were due to carcinoma, and a quarter were due to cystic hyperplasia. Firm fibroadenoma was next in frequency, followed by fat necrosis and chronic inflammation.

TABLE I.

Diagnosis of 100 Consecutive Breast Lumps Seen at Operation in Sydney Hospital from January 1, 1956, to July 1, 1957.

Types of Breast Lumps.	Number of Cases.	Cases with No Lump.[1]
Neoplasms:		
Firm fibroadenoma	7	—
Soft fibroadenoma	0	—
Cystosarcoma phyllodes	1	—
Duct papilloma	2	1
Duct carcinoma	1 (Comedo)	—
Infiltrating carcinoma:		
Scirrhous	45	5[2]
Medullary	2	—
Papillary	0	—
Mucoid	1	—
Rare forms	0	—
Paget's disease of the nipple	0	1
Sarcoma	0	—
Rare tumours	2 (Lipomata)	—
Dysplasia:		
Cystic hyperplasia, Fibrosing adenosis	26	—
Lactating nodule	2	—
Duct ectasia	2	—
Trauma:		
Fat necrosis	5	—
Hæmatoma	0	—
Infection:		
Chronic inflammation and abscess	4	—
Tuberculosis, etc.	0	—
Total	100	7

[1] Cases admitted during the same period in which there was no lump.
[2] The five cases of scirrhous carcinoma had had excisional biopsy elsewhere.

Clinical Diagnosis.

Table II compares and contrasts the clinical features of the common or important breast lumps, which are as follows.

Firm Fibroadenoma.

Firm fibroadenoma (pericanalicular fibroadenoma) frequently forms between puberty and the age of 25 years. The tumour is seldom painful or tender, is most commonly found in the upper half of the breast, and when first noticed is often one to two centimetres in diameter. It is spherical or ovoid in shape, with a smooth contour and very well-defined edge; the consistency is firm and elastic. Excessive mobility is a characteristic feature, which Atkins (1951) has aptly likened to that of a piece of ice in a tumbler. Attachment is absent, not only from the skin, nipple and deeper structures, but to a surprising extent from the breast tissue itself. This is the easiest lump in the breast to diagnose correctly on clinical grounds; yet, as will be seen later, diagnostic errors may occur. It should be removed.

[1] Based on a paper read at Sydney Hospital Refresher Week, September 9, 1957.

Soft Fibroadenoma.

Soft fibroadenoma (intracanalicular fibroadenoma) rarely forms before the age of 35 years. The history is one of rapid growth of a painless lump in the breast. The tumour tends to be deep, is rounded and lobulated and has a very definite edge and a soft consistency. Again, mobility is marked and attachment absent. The features are identical with those of lipoma and *cystosarcoma phyllodes*. Growth tends to continue, and if the tumour is not removed it may become very large and may even fungate through the skin.

Cystosarcoma Phyllodes.

Cystosarcoma phyllodes results from an alteration in the stroma of a fibroadenoma. The clinical features are those of soft fibroadenoma. The lesion tends to enlarge rapidly, and if it is not removed it may attain giant size and even ulcerate through the skin. Occasionally, it behaves as a sarcoma and metastasizes to lungs, lymph nodes, etc.

Duct Papilloma and Duct Carcinoma.

Duct papilloma and duct carcinoma are often clinically indistinguishable, and may or may not cause a lump. The maximal age incidence is 35 to 50 years. Nipple discharge is present in most cases, and is blood-stained or sometimes merely serous. The lump, if it is palpable, is soft and situated deep to the nipple or areola; emerging from this there may be a sector-shaped area of induration. Finger-tip pressure clockwise around the edge of the areola may reveal the site of the tumour and the source of the discharge. Transillumination may localize an impalpable lesion.

TABLE II.

Symptoms and Physical Findings Associated with Common Lumps in the Breast.

Clinical Features.	Firm Fibro-adenoma.	Soft Fibro-adenoma.	Scirrhous Carcinoma.	Medullary Carcinoma.	Cystic Hyper-plasia.
Subject's age in years.	14 to 25	Over 35	40 to 60	25 to 35	30 to 50
Pain	—	—	Rare.	Rare.	Frequent.
Nipple discharge	—	—	Occasional.	Occasional.	Sometimes.
Recent nipple retraction.	—	—	Late.	—	—
Position in the breast.	Upper half.	Deep.	Upper outer quadrant.	Central.	Upper outer quadrant.
Size	One to two centi-metres.	Larger.	Varies.	Large.	Varies.
Shape	Spherical or ovoid.	Spherical or ovoid.	Irregular.	Rounded.	Irregular.
Contour	Smooth.	Lobulated.	Irregular.	Smooth.	Irregular.
Edge	Definite.	Definite.	Indefinite.	Definite.	Indefinite.
Consistency	Firm.	Soft.	Hard.	Soft.	Firm.
Mobility	+++	+++	±	+	+
Attachment	—	—	±	—	—
Tenderness	—	—	Rare.	Rare.	Frequent.
Translucency	—	—	—	—	±
Other breast tissue.	—	—	—	—	Nodular.
Axillary nodes	—	—	±	±	±

Scirrhous Carcinoma.

Scirrhous carcinoma seldom causes pain, except in advanced cases. However, Lewison (1955) emphasizes that there may be pain and tenderness in early cases, when the pain resembles that of cystic hyperplasia, with premenstrual exacerbation. Nipple discharge may be present, and it is blood-stained, serous or, less often, milky. The nipple may become deviated, or broadened and flattened or, in later cases, retracted and perhaps elevated. This recent retraction must be distinguished from simple inversion of the nipple, which may be unilateral or bilateral, has usually been present for many years and usually

allows the nipple to be pulled out from its sulcus. The lump of carcinoma is most commonly situated in the upper and outer quadrant of the breast, is irregular and has no definite edge, merging into the surrounding breast tissue. It is hard and, unlike the lump of cystic hyperplasia, is easily felt when it is compressed by the flat of the fingers against the chest wall. There may be dimpling and, later, fixation of the skin over the tumour. *Peau d'orange*, nodules and ulceration are late skin changes. The tumour may be attached to the underlying fascia and muscles, with reduction in mobility when the *pectoralis major* is made to contract.

Retraction phenomena and alteration of breast contour may be made more apparent when the breast is inspected with the patient sitting in the following positions: first, with the arms by the sides; secondly, as the arms are slowly raised above the head; thirdly, with the patient leaning well forwards so that the breasts swing away from the body; and fourthly, with the hands on the hips, the pectoral muscles first being relaxed, then contracted. Simply moving the breast from side to side, with the patient supine, may accentuate skin dimpling, as may the "plateau test", in which the flat of the hands placed on either side of the lump are gently pressed together.

Medullary Carcinoma.

Medullary carcinoma has a maximal age incidence of 25 to 35 years. Frequently, conspicuous veins are seen in the skin overlying the tumour, which is usually deep in the central part of the breast. It is of relatively rapid growth and large size, often being four to six centimetres in diameter when first examined. It is rounded, has a definite edge and is much softer than a scirrhous carcinoma. There is not the same tendency to adhere to the skin, although fungation may eventually occur. Axillary node involvement is late.

Infiltrating Papillary Carcinoma and Mucoid Carcinoma.

These resemble medullary growths in their circumscription, soft consistency and lack of fixation. They may attain considerable size before metastasis occurs.

Paget's Disease of the Nipple.

Paget's disease of the nipple usually commences in patients over 50 years of age. Initially there is an intractable eczema of the nipple, but sooner or later, usually within two years but occasionally not till after 10 years, a carcinoma appears in the breast, frequently at some distance from the nipple.

Sarcoma.

Sarcoma is difficult to differentiate from medullary carcinoma, but it is even softer, and its growth is more rapid.

Cystic Hyperplasia.

Cystic hyperplasia, in its diffuse form, causes a nodularity in part or whole of one or both breasts. However, it is the localized form, in which there is a definite lump, which concerns us.

The great majority of cases occur in patients between the ages of 30 and 50 years. Usually about the menopause the symptoms, and later the signs, disappear. Pain, tenderness and the size of the lump commonly increase in the seven to 10 days before a menstrual period, and regress on the first or second day of the period. Nipple discharge is sometimes present and it may be serous, blood-stained or dark green in colour. The lump, like that of carcinoma, is commonest in the upper and outer quadrant of the breast. It is irregular (sometimes sector-shaped) and has an ill-defined edge. The consistency is firm. A swelling which can be clearly felt when it is compressed against the chest wall with the flat of the fingers, is almost certainly not due to cystic hyperplasia. The lump is attached to the breast; attachment to deep structures never occurs and to the nipple only occasionally. A lump attached to the skin should always suggest carcinoma rather than cystic hyperplasia. Transillumination may help in some cases. Fat, cysts filled with clear fluid, and normal breast tissue are translucent; tumours and blood-filled cysts are opaque; areas of fibroadenosis are intermediate. If a lump is due to cystic hyperplasia there is nearly always nodularity elsewhere in the same breast and probably in the other breast; this is a most important diagnostic point. The axillary nodes are often enlarged, and they are firm, mobile, discrete and tender.

The cause of the lump in the breasts of women over 40 years of age is a cyst or a group of cysts, in young people it is an area of fibroadenosis. When a cyst lies in relatively normal breast tissue, it is often possible to identify it clinically. Its surface is smooth and its consistency varies according to the tension within. When the cyst is firmly placed against the chest wall with one hand, it is often possible to feel its tense, elastic nature with a finger of the other hand. If, as is more usual, the cyst is surrounded by an area of fibroadenosis, its presence is much more difficult to detect.

Aspiration, even in the doctor's rooms, is sometimes used, but it is seldom satisfactory, either in diagnosis or in treatment. The needle may fail to find the cyst, or clear fluid may be withdrawn from a cyst adjacent to an early carcinoma.

When there is a local lump due to cystic hyperplasia, the clinical diagnosis must be uncertain, and exploration becomes a necessity.

Fibrosing Adenosis.

Fibrosing adenosis is a variant of cystic hyperplasia, and produces a firm lump with an indefinite edge. It may mimic cancer, and is the commonest cause of an unnecessary radical mastectomy for a benign condition. Even the histological picture may resemble that of carcinoma.

Mammary Duct Ectasia.

In mammary duct ectasia the ducts become distended with inspissated secretion, and periductal inflammation produces a mass with retraction signs. This lesion may simulate cancer so closely that it has led Haagensen (1956) to state that "it is never safe to perform radical mastectomy without microscopic proof of the nature of the lesion".

Fat Necrosis.

Fat necrosis is easily mistaken for carcinoma, and usually affects stout, middle-aged women. A history of trauma is obtained in 50% of cases. After a knock, a surgical operation, a subcutaneous infusion, or even indirect violence such as contraction of the pectoral muscles, a lump, often painless, appears. Ecchymosis may be present, in which case it is the most striking feature of the lesion. In some patients in whom there is no ecchymosis the skin over the lesion is reddened. Pain is often absent. The nipple is occasionally retracted or deviated. The lump is usually in the superficial adipose tissue, close to the skin, the commonest site being the areolar region. It is usually small, about two centimetres in diameter, rounded, and with a fairly definite edge. The consistency is hard, and the swelling is usually attached to the overlying skin, which is retracted.

Chronic, Non-Specific Inflammation and Abscess.

Chronic, non-specific inflammation and abscess are often difficult to diagnose, and they can be distinguished from carcinoma only by exploration.

It will be realized that a number of benign lesions of the breast (e.g., fat necrosis, duct ectasia, fibrosing adenosis and low-grade abscess) may closely simulate carcinoma. Conversely, the circumscribed type of carcinoma may seem to be as well delimited as a cyst; and the surgeon is seeing earlier cases of breast cancer, which may not show one of the cardinal signs. Table III indicates the clinical diagnosis made in some of the pathological conditions which caused the 100 lumps under consideration. It will be seen that fat necrosis and chronic, non-specific inflammation and abscess gave great difficulty in diagnosis. Difficulties were encountered even in cases of fibroadenoma, particularly when the lesion was surrounded by a zone of fibroadenosis.

Breast Biopsy.

In view of the pitfalls of clinical diagnosis, breast biopsy and histological examination (preferably by frozen section) are the ideal in many cases, before any therapeutic procedure (particularly radical mastectomy) is carried out. The theoretical danger of preliminary biopsy in cancer cases is the dissemination of malignant cells in the blood and lymph streams. Reliable evidence of the effect of biopsy on survival is almost impossible to obtain. However, biopsy is now regarded as a prudent and justifiable risk.

Opinions differ widely as to the relative merits of incisional and excisional biopsy. In the former procedure the tumour is incised *in situ*, and a small wedge is removed for examination. This method reduces operative interference and bleeding to a minimum, and is supposed thereby to reduce the risk of metastasis. It is certainly the method of choice for large

tumours or for those which appear obviously malignant. In excisional biopsy the whole swelling is removed; it may be inspected, carefully palpated and cut across in a number of places, a piece being removed for histological examination. Of course the excision is unlikely to be clear of the zone of microscopic infiltration by malignant cells. This method is favoured by many surgeons and is particularly applicable when the tumour is small or appears obviously benign. The arguments in support of these different procedures could not be proved without analysis of very many cases with complete

TABLE III.

Comparison of Clinical Diagnosis and Pathological Findings in Some Breast Lumps Seen at Operation in Sydney Hospital.

Pathological Diagnosis.	Number of Subjects.	Clinical Diagnosis.	Number of Subjects.
Firm fibroadenoma ..	7	Fibroadenoma	5
		Cystic hyperplasia ..	1
		Doubtful lump	1
Scirrhous carcinoma ..	45	Scirrhous carcinoma ..	33
		Lump	10
		Cystic hyperplasia ..	1
		Fibroadenoma	1
Cystic hyperplasia ..	26	Cystic hyperplasia ..	11
		Doubtful lump	14
		Fibroadenoma	1
Fat necrosis	5	Probable carcinoma ..	1
		Lump	3
		? Phlebitis	1
Chronic inflammation and abscess	4	Carcinoma	1
		Inflammation	1
		Lump	2

follow-up investigations, and even then the answer would probably not carry conviction. Simple mastectomy must never be performed as an alternative diagnostic measure to biopsy; the subsequent radical mastectomy has disappointing end results.

Table IV illustrates the part which breast biopsy played in the management of the 100 cases under review.

In 26 cases, the clinical diagnosis of carcinoma was made with such certainty that mastectomy without preliminary biopsy was carried out. In no case was radical mastectomy carried out

TABLE IV.

Table to Illustrate the Role of Breast Biopsy in the Management of 100 Consecutive Breast Lumps Seen at Operation in Sydney Hospital.

Clinical Diagnosis.	Management.	Benign Lesion.	Carcinoma.	Diagnosis Made on Gross Features.	Diagnosis Made on Frozen Section.
Carcinoma.	Mastectomy.	—	26	—	—
Doubtful.	Biopsy and local excision.	34	—	15	19
Doubtful.	Biopsy and mastectomy.	—	21[1]	6	14
Benign.	Exploration and local excision.	17	—	17	—
Benign.	Biopsy and mastectomy.	—	2	1	1

The last two columns show the basis for the decision as to the type of operation to be performed.

[1] One lump diagnosed on paraffin section; mastectomy performed seven days later.

for a benign condition. However, the latter error is by no means unknown; in fact, Yeates (1953) found that radical mastectomy had been performed no less than 16 times on the 199 patients with cystic hyperplasia admitted to Sydney Hospital during the ten-year period ending June 30, 1952. Perhaps the availability of frozen section examination in recent years accounts for the new freedom from this error.

In 55 cases, the clinician had some doubt as to the nature of the lump; 34 of these lumps were ultimately found to be benign and 21 malignant. By the use of gross examination of the lesion at biopsy, frozen section, and, in one instance, examination of a paraffin section, the correct diagnosis and treatment were achieved in each case.

In 19 cases a definite diagnosis of some benign condition was made. However, in two of these cases the lump was in fact a carcinoma; but the day was saved in each instance by breast biopsy, relying on the gross features once and on the frozen section once; on each occasion immediate radical mastectomy was performed. Thus, even in the case of lumps which appear obviously benign, the surgeon should be prepared to carry on with the radical operation if necessary. Before operation, the patient's permission is obtained and blood is made available.

Gross Examination.

The gross features of several lesions are summarized in Table V.

The lump of scirrhous carcinoma is hard, stands up to the knife and does not tend to roll about under the blade like the lump of cystic hyperplasia. It cuts like an unripe pear (with a gritty sensation), or like a raw potato. It may be heard to grate or creak while being cut. Usually both cut surfaces are found to be concave, in contrast to the flat surface of cystic hyperplasia and the bulging surface of fibroadenoma. The edge is indefinite and irregular, with radiating projections into the surrounding tissue, the result being a puckered appearance. There is no suggestion of a capsule, and it is impossible to separate the tumour from the breast. This contrasts with fibroadenoma,

TABLE V.

The Gross Features of Some of the Commoner Breast Lumps.

Clinical Features.	Firm Fibro-adenoma.	Soft Fibro-adenoma.	Scirrhous Carcinoma.	Medullary Carcinoma.	Cystic Hyper-plasia.
Consistency ..	Very firm (like cartilage).	Soft.	Hard (like unripe pear).	Soft.	Tough (like india-rubber).
Shape of cut surface.	Bulging.	Bulging.	Concave.	Bulging.	Flat.
Edge	Definite.	Definite.	Indefinite.	Definite.	Indefinite.
Capsule	Present.	Present.	Absent.	Absent (microscopically).	Absent.
Colour of cut surface.	Whitish.	Grey.	Grey. (yellow spots or streaks).	Greyish-white.	White or yellow.
Texture and markings.	Granular with whorls or splits; may be tiny cysts.	Moist and succulent concentric clefts, like a cut cabbage.	Gritty specks; may be little hair-like processes.	Frequently hæmorrhage and cysts; friable.	Pink and grey dots; cysts (may be blue domes).

which, when incised, tends to pop out from its sheath, to which, however, it is often attached at one point. In scirrhous carcinoma, the colour of the cut surface is definitely grey, and its appearance has been likened to the interior of an unripe pear; here and there yellowish, chalky streaks or spots are present. When the cut surface is scraped, small hair-like processes may stand up, as if a piece of wood had been scraped against the grain.

Reference to Table IV shows that, in the 74 cases in which biopsy was used, reliance was placed on gross examination alone on 39 occasions without error. In 34 cases, examination of a frozen section was employed, but probably this was confirmatory in a number of instances, the gross features having already given a fairly definite indication of the diagnosis. The macroscopic features of the lump at biopsy thus provide valuable diagnostic evidence, and are worthy of careful study.

Frozen Section.

Frozen section examination allows the surgeon to receive the histological report while the operation is still proceeding, usually within 10 to 15 minutes of taking the biopsy. By this means, the patient may be spared the need for two operations and two anæsthetics, with the additional danger, anxiety and expense. The tissue may be cut, stained and mounted for microscopic examination within a minute or two of its receipt. The sections are uniform and thin, and with the use of the polychrome methylene blue stain the tissues appear to be demonstrated as clearly as with routine hæmatoxylin and eosin preparations

made after 24 hours' fixation. The pathologist must be skilled in the method and must be willing, whenever necessary, to admit uncertainty. The surgeon must accept the pathologist's occasional refusal to make a diagnosis in a doubtful case.

Table IV shows that frozen section examination was used on 34 occasions. In every case the diagnosis was correct, confirmed by subsequent paraffin sections. Frozen sections were found to be an invaluable aid; ideally they should be available in the management of every breast lump.

Paraffin Section.

Paraffin sections may be used if a frozen section is not available, and in the occasional case in which frozen section examination results in uncertainty. The surgeon must never undertake radical mastectomy without convincing evidence of malignancy. If the time lag between breast biopsy and definitive surgery is merely a matter of several days, the delay, although undesirable, has not been shown to prejudice ultimate survival.

During the period under review, five patients were transferred to Sydney Hospital in whom breast biopsy performed elsewhere had revealed scirrhous carcinoma. The average interval between biopsy and definitive surgery was seven weeks. Prolonged delays, due to awaiting the result of sections, arranging the transfer of patients to another hospital, etc., had indeed become excessive. Of course breast biopsy should be done only where there are facilities for performing radical mastectomy, either immediately or, in doubtful cases, after a prompt report on paraffin sections.

Summary.

The records of a small series of cases of lumps in the breast have been reviewed, in order to illustrate the difficulties of clinical diagnosis, and the place of biopsy in their management.

Whenever biopsy is done, the surgeon should be prepared for radical mastectomy.

The gross features found at biopsy are worthy of careful study.

Frozen section examination is a valuable aid, and ideally should always be available.

On occasions, the diagnosis must await the result of examination of paraffin sections, any necessary definitive surgery being carried out a few days later.

Acknowledgements.

I wish to thank Dr. A. A. Palmer and Dr. E. Hirst, of the Kanematsu Institute of Pathology, for their aid.

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ARE THERE TWO OXYTOCIC HORMONES?

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ALTHOUGH oxytocin was synthesized in 1953 by du Vigneaud, Ressler, Swan, Roberts, Katsoyannis and Gordon, it was not assayed in the body fluids until the following year, when Bisset and Walker reported finding it in rat blood. Using a modification of their method for extracting it from blood, we have assayed oxytocin in women during non-pregnancy, pregnancy, labour and lactation (Hawker and Robertson, 1957*a*, 1957*b*; Hawker, 1957) and in cows and goats before and during milking (Hawker and Roberts, 1957). With a different extraction method we have extracted oxytocin from the hypothalami of cats, rats, mice and cows (Robertson and Hawker, 1957).

The test we have applied to identify oxytocin in the extracts is that initially described by Van Dyke, Chow, Green and Rothen (1942) and by Ames and Van Dyke (1952), who found that thioglycollic acid inactivated the posterior pituitary hormones. All the extracts of blood and hypothalami that we prepared showed residual oxytocic activity after treatment with thioglycollic acid and hence possessed an oxytocic activity that could not be due to oxytocin. The oxytocic activity of the extracts was assayed on the isolated sensitized uterus of virgin Wistar strain rats as described by Holton (1948) and by Gaddum, Peart and Vogt (1949). By oxytocic activity is therefore meant the uterine-stimulating activity of the extracts.

A number of substances are known which stimulate the rat uterus to contract, and besides oxytocin these include potassium, acetylcholine, 5-hydroxy-tryptamine, bradykinin, hypertensin (angiotonin), vasopressin (antidiuretic hormone), pepsanurin, pepsitocin, pain-producing substance, kallidin, substance P, substance Z, substance U, pain-producing substance (PPS) and pepsitensin: perhaps there are other substances, and some of these may or may not be present in our extracts.

From a series of biological, chemical and pharmacological experiments conducted in an attempt to determine some of the properties of this unidentified oxytocic substance in our blood and hypothalamic extracts, the following observations on it were made.

1. It is retained during dialysis by a "Cellophane" membrane through which oxytocin dialyses.

2. Its dose response curve when tested on the virgin rat uterus is not significantly different from that of "Pitocin".

3. It is not recoverable in significant amounts if the hypothalamus is left in the animal for 30 minutes after death.

4. It is destroyed by chymotrypsin at pH 7·3 and 37° C., and by an enzyme in pregnancy plasma at pH 7·5 (Hawker, 1956); it is not inactivated by trypsin (pH 7·3) or pepsin (pH 2·5).

5. It is stable at 100° C. in faintly acid solutions, but is easily destroyed by alkalis or strong acids.

6. It is stable when stored in the deep-freeze at either pH 7·5 or pH 2 for at least four months.

7. It is equiactive when tested on the rat uterus and guinea-pig and rabbit ileum, and its action is not blocked by atropine, mepyramine, dibenamine or lysergic acid diethylamide.

8. It does not induce milk ejection when assayed on the mammary gland of lactating rabbits.

9. It is not potassium.

10. It is not thought to be a pain-producing substance (PPS) as it is present in blood collected in silicone coated syringes and is extracted in similar coated glassware.

As a result of these investigations it can reasonably be concluded that oxytocic substance is not oxytocin, but that in many chemical and pharmacological respects it closely resembles the posterior pituitary oxytocic hormone. Although its identity is unknown, the problem has become so resolved as to suggest that it is bradykinin, kallidin, substance U, substance Z or an unknown polypeptide with a molecular weight larger than oxytocin (1000) but with closely similar properties. The possibility therefore emerges that two oxytocins are present in the hypothalamus and blood in man and in other species.

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[1] Read at a meeting of the surgical staff of the Brisbane Hospital on October 10, 1957.

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REVIEW OF 1094 OPERATIONS FOR PEPTIC ULCER.[1]

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DURING the five-year period ending June 30, 1957, 1094 operations were performed at the Brisbane Hospital for benign peptic ulcer. This total included 311 operations for closure of perforated ulcer and 783 other operations for ulcer. Altogether there were 816 males and 278 females, a male predominance of 2·9 to one. As the points of interest about perforations are rather different from the other cases, we will discuss them separately.

OPERATIONS FOR PEPTIC ULCER, EXCLUDING PERFORATIONS.

This group includes 681 elective operations for peptic ulcer and 102 emergency operations for bleeding peptic ulcer. The majority of these operations were partial gastrectomies, but there were numerous other procedures carried out (Tables I and II). The 681 partial gastrectomies included 580 of the Pólya type and 101 of the Billroth I type.

TABLE I.
Elective Operations.

Operation.	Number of Patients.	Number of Deaths.	Mortality Percentage.
Partial gastrectomy	592	15	2·5
Gastro-enterostomy	48	1	2·1
Gastro-enterostomy followed one week later by partial gastrectomy because of stomal obstruction	1	1	—
Gastro-enterostomy *plus* vagotomy	25	0	—
Partial gastrectomy *plus* vagotomy	5	0	—
Vagotomy	2	0	—
Pyloroplasty	2	0	—
Pyloroplasty *plus* vagotomy	1	0	—
Excision of ulcer	3	0	—
Gastrotomy	1	0	—
Drainage of abscess around missed perforation	1	1	—
Total	681	18	2·6

This paper does not attempt to give the long-term results of the various operations but rather the indications for operation, the position of ulcers, the age and sex incidence and the immediate morbidity and mortality rates.

The indications for surgical intervention included failure of medical treatment (persistent pain or persistent bleeding), fear of malignancy and pyloric obstruction. A history of previous perforation or bleeding was a less absolute indication. The majority of patients who underwent elective surgery complained of persistent pain, this sometimes being the only indication for surgery, but in other cases a history of previous bleeding and/or perforation was also present. A history of previous perforation was present in 102 cases or 13% of the total. It is interesting to note that in the same five-year period there were 311 operations for closure of perforated ulcer. As the vast majority of perforated ulcers in this hospital are treated surgically, and have been for many years, these figures indicate that about one perforated ulcer in three will come to further surgery. There was a history of hæmatemesis and/or melæna, past or present, in 40% of cases. In almost one-third of these continued bleeding necessitated an emergency operation.

Patients admitted to the Brisbane Hospital with hæmatemesis and/or melæna are in the first instance admitted to medical wards, and, for the most part, conservative treatment is successful. However, should bleeding continue after medical treatment is instituted, the patient is examined by a surgeon. Continued or repeated bleeding, a history of previous bleeding, length of ulcer history, age, general condition and associated pain are taken into account in making a decision on subsequent treatment. In all, there were 102 emergency operations for bleeding during this five-year period. In this same period there were approximately 1250 admissions to hospital for bleeding from suspected or proven peptic ulcer. So it can be seen that the great majority of bleeding ulcers are treated conservatively in this hospital. In other words, an attempt is made to select for surgical treatment those patients who will probably die with continued medical treatment. Inevitably, mistakes are made in both directions, but the results on the whole are probably better than the routine treatment of all bleeding ulcer patients on either exclusively medical or exclusively surgical lines. In the 102 emergency operations for bleeding there were 24 deaths, a mortality rate of 23·5%. Some of these patients were extremely "poor risks", as can be judged from some of the types of operation performed (Table II). The 89 patients on whom an emergency gastrectomy was performed were probably slightly better surgical "risks", but there were 16 deaths, a mortality rate of 18%.

TABLE II.
Emergency Operations.

Operations.	Number of Patients.	Number of Deaths.	Mortality Percentage.
Partial gastrectomy	89	16	18·0
Gastro-enterostomy	1	1	—
Gastro-enterostomy *plus* ligation of vessels, followed one week later by partial gastrectomy because of afferent loop obstruction	1	0	—
Sleeve resection	1	1	—
Ligation of vessels and excision of ulcer	5	2	—
Ligation of vessels *plus* excision of ulcer, followed by partial gastrectomy 10 days later because of perforation of ulcer	1	1	—
Gastrotomy *plus* ligation of vessels	4	3	—
Total	102	24	23·5

TABLE III.
Type of Ulcer.

Type of Ulcer.	Number of Patients.	Males.	Females.	Male : Female Ratio.	Remarks.
Gastric.	307	190	117	1·6 : 1	32 patients had multiple gastric ulcers.
Duodenal.	400	330	70	4·7 : 1	
Both gastric and duodenal.	30	22	8	2·7 : 1	
Stomal.	26	20	6	3·3 : 1	20 after gastro-enterostomy and 6 after partial gastrectomy.
Pyloric.	20	15	5	3·0 : 1	

Fear of malignancy is frequently an indication for operation on gastric ulcers. This is reflected in the relatively high proportion of gastric to duodenal ulcers in this series, namely, 307 gastric to 400 duodenal ulcers (Table III). Most authorities state that the incidence of duodenal ulcer is much higher than that of gastric ulcer, but whereas there is no risk of malignant change in a duodenal ulcer there is about a 6% chance of a chronic

[1] Read at a meeting of the surgical staff of the Brisbane Hospital on October 10, 1957.

TABLE IV.
Age Incidence.

Age in Years.	Total Number of Patients.	Total Number of Deaths.	Mortality Percentage.	Emergency Operations.		
				Number of Patients.	Number of Deaths.	Mortality Percentage.
10 to 19	3	0	Nil	2	0	Nil
20 to 29	39	3	7·7	6	2	33·3
30 to 39	101	1	1·0	7	0	Nil
40 to 49	214	5	2·3	18	3	16·6
50 to 59	221	8	3·6	22	2	9·1
60 to 69	148	17	11·5	30	11	36·6
70 to 79	52	8	15·4	16	6	37·5
80 to 89	5	0	Nil	1	0	Nil
Total	783	42	5·4	102	24	23·5

gastric ulcer becoming malignant. Surgical consultations are therefore much more readily sought in cases of persistent gastric ulcer than in duodenal ulcers that fail to respond to medical treatment.

In 133 cases there was clinical, radiological or operative evidence of pyloric obstruction, an incidence of 17%. Most of these patients were treated by partial gastrectomy, but on some, particularly the elderly ones in poor condition, a gastro-enterostomy was performed.

TABLE V.
Complications in Survivors.

Complication.	Number of Patients.
Chest, including respiratory and cardio-vascular complications	40
Wound infection	31
High gastric aspirations in early post-operative period	14
Post-operative hæmatemesis and/or melæna	9
Burst abdomen	9
Gastric or duodenal fistula	10
Urinary infection	2
Deep vein thrombosis in lower extremity	4
Intraperitoneal hæmorrhage	1
Pelvic collection	2
Peritonitis	5
Paralytic ileus	4
Foot drop	1
Uræmia	2
Intestinal obstruction	1
Axillary artery embolus	1
Incarcerated hernia	1
Jaundice	5
Diarrhœa	3
Total	145

Age Incidence.

The majority of peptic ulcer patients who come to surgery do so in middle life, often having had symptoms for many years. The maximum incidence in this series is in patients aged between 40 and 60 years. As can be seen from Table IV, emergency operations for bleeding reach a maximum incidence in a later age group. In patients aged over 60 years, 23% of the operations performed were emergency operations for bleeding, whereas in those aged under 60 years only 9% of the operations were emergencies. As might be expected, the mortality rate for emergency operations is much higher in the later age groups, namely, 37% in the patients over 60 years of age and 13% in the patients under 60 years of age.

Another interesting point emerging from these figures is that there were two deaths in six emergency operations on patients in the 20 to 29 years age group. Both were female patients aged 24 years, with gastric ulcers, and both patients died a few hours after operation from peripheral circulatory failure. In both cases gastrotomy and ligation of bleeding vessels were performed. There is a tendency to be very conservative in the case of young patients with bleeding peptic ulcer, and at times the surgeon is called too late.

Sex Incidence.

In this series there were 563 males to 220 females, which is a male preponderance of 2·5 : 1·0. This male preponderance, although universal, varies from 6·0 : 1·0 to 1·1 : 1·0 in published figures. The male predominance was more marked in duodenal than in gastric ulcers, being 4·7 : 1·0 in the former and 1·6 : 1·0 in the latter.

Complications in Survivors.

Among the 741 survivors in this series there were 145 complications worthy of mention (Table V). In some instances the patient's post-operative stay in hospital was considerably prolonged, but only 92 or 12·4% of the survivors were in hospital for three weeks or more. In 500 cases or 67·4% of the total, the post-operative stay in hospital was two weeks or less.

Deaths.

There were 42 deaths in 783 operations, which is a mortality rate of 5·4%. These totals included 102 emergency operations for bleeding, with 24 deaths. Tables I and II indicate the types of operations performed, the number of deaths associated with them and the mortality rates. Table VI lists the causes of death and also indicates the causes of death in emergency operations.

TABLE VI.
Causes of Death.

Causes of Death.	Number of Deaths.	Number of Deaths in Emergency Operations.
Chest complications	12	7
Cerebral thrombosis	1	0
Continued bleeding, ? blood dyscrasia	1	0
Cardiac arrest	1	1
Post-operative shock	4	4
Leak from duodenal stump	11	7
Leak from gastric anastomosis	7	3
Peritonitis from leaking anastomosis of transverse colon (in this case the ulcer was adherent to the transverse colon and was thought to have been malignant)	1	0
Bleeding from duodenal ulcer remnant	1	1
Intraperitoneal hæmorrhage	1	1
Plastic peritonitis and intestinal obstruction	1	0
Peritonitis after drainage of abscess around missed perforation	1	0
Total	42	24

An interesting point emerging from these figures is that there was a leak from the duodenal stump or gastric anastomosis in 28 patients, with 18 deaths, which indicates what a lethal complication this is. This complication occurred 11 times in 89 emergency gastrectomies, that is, 12·5%, and 17 times in 592 elective gastrectomies, that is 3%; or in other words, a leaking anastomosis or duodenal stump is four times more likely to occur in emergency gastrectomies than in elective gastrectomies. This, no doubt, is mainly due to the inflamed and œdematous tissues frequently encountered around bleeding ulcers.

Operations for Closure of Perforated Ulcer

At this hospital, the vast majority of perforated ulcers are treated by operation. Conservative measures are employed when the clinical signs point to a small leak which is localizing or in obviously moribund patients. However, we are reviewing only the surgically treated cases, as it is difficult to be certain of the diagnosis in all cases in which a small leak is presumed to be present.

Sex Incidence.

In this series of 311 patients there were 253 males and 58 females, which is a ratio of 4·3 to one. This figure closely corresponds with the ratio quoted by several authors overseas.

Age Incidence.

The maximum incidence of perforation was in the fifth decade, but it was not uncommon at any age from 30 to 70 years. The age of the patient has a most important influence on the mortality rate, there being no deaths in patients aged under 40 years, but there was a steadily increasing mortality rate thereafter (Table VII).

TABLE VII.
Perforated Ulcers: Age Incidence.

Age in Years.	Number of Patients.	Number of Deaths.	Mortality Percentage.
10 to 19	3	0	Nil
20 to 29	30	0	Nil
30 to 39	64	0	Nil
40 to 49	87	4	4·6
50 to 59	57	5	8·7
60 to 69	48	7	14·5
70 to 79	20	8	40·0
80 to 89	1	1	100·0
90 to 99	1	1	100·0

Position of Ulcer.

In this series of perforations there were 215 perforated duodenal ulcers and 88 perforated gastric ulcers (Table VIII).

The predominance of duodenal ulcers in operations for perforation was more striking than in the elective operations for peptic ulcer, in which relatively more gastric ulcers were encountered. This is probably because gastric ulcers are more likely to be resected on account of the risk of malignancy. It is interesting to note that the male predominance was not nearly

TABLE VIII.
Perforated Ulcers: Type of Ulcer.

Type of Ulcer.	Number of Patients.	Males.	Females.	Ratio of Males to Females.
Gastric	88	57	31	1·8:1
Duodenal ..	215	188	27	7·0:1
Pyloric	5	5	—	—
Stomal	3	3	—	—

so great in cases of perforated gastric ulcer (1·8 to one) as it was in cases of perforated duodenal ulcer (7·0 to one).

Perforation occurred in three stomal ulcers. One of these ulcers was a sequel to gastro-enterostomy and the other two followed gastrectomy.

Deaths.

There were 26 deaths from perforated ulcers, including 16 patients with duodenal and 10 patients with gastric ulcers. This corresponds to a mortality rate of 8·3%. The mortality rate was 11·3% for gastric ulcer and 7·4% for duodenal ulcer. The higher figure for gastric ulcer is no doubt due to the fact that perforations of gastric ulcers tend to be larger than such lesions in the duodenum.

The interval of time elapsing between perforation and operation is a most vital factor. The majority of patients had an operation within 12 hours of the onset of perforation, and the number of deaths in this group was small in proportion to the number of deaths when the time interval was over 12 hours (Table IX).

Age, *per se*, has a most significant influence on the prognosis. There were 241 patients aged under 60 years, nine of whom died (that is, 3·7%), whereas there were 70 patients aged 60 years or over, of whom 17 died (that is, 24%).

TABLE IX.
Time Interval.

Time Interval Between Perforation and Operation in Hours.	Number of Patients.	Number of Deaths.	Mortality Percentage.
1 to 6 ..	86	3	3·5
6 to 12 ..	160	7	4·4
12 to 24 ..	44	6	13·6
Over 24 ..	21	10	47·6

The majority of deaths were due to peritonitis occurring in late cases, or in patients aged over 60 years. The various causes of death are listed in Table X.

TABLE X.
Cause of Death.

Causes of Death.	Number of Deaths.
Peritonitis and ileus	14
Subphrenic abscess and duodenal fistula	1
Small gut obstruction and fistula formation	1
Hæmorrhage from ulcer	1
Peripheral circulatory failure	5
Uræmia	2
Congestive cardiac failure	2
Total	26

Summary.

In the five-year period ending June, 1957, 1094 elective and emergency operations were performed for peptic ulcer, with an over-all mortality of 6·2%.

There were 311 perforated peptic ulcers treated surgically, with a mortality of 8·3%.

Elective gastrectomies were performed on 592 cases of benign peptic ulcer, with a mortality of 2·5%.

Acknowledgement.

We should like to thank Dr. A. D. D. Pye, General Medical Superintendent of the Brisbane Hospital, for permission to publish these figures.

Reviews.

Mental Health and Education in Hong Kong. By K. E. Priestley, M.A. (Oxon.), and Beryl R. Wright, B.Ec. (Sydney); 1956. London: Geoffrey Cumberlege, Oxford University Press. 7¼″ × 5″, pp. 104. Price: 14s. 3d.

THIS booklet consists of four lectures by Professor K. E. Priestley and two by Miss Beryl Wright, delivered in the first place as an extramural series conducted by the University of Hong Kong. The final chapter, produced jointly by the authors, records group discussions based on the lectures, with the answers given to questions arising from these discussions. The lectures were widely supported by the public, and audiences included teachers, doctors, social workers, nurses, and members of police and prisons services.

The publication is the recording of a sincere attempt on the one hand to foster closer relations between the university and the general public, and on the other to make a planned attack on the question of mental health. The criteria used for defining the mental health of an individual are those commended by Dr. Fremont-Smith and Dr. Rees, of the World Federation for Mental Health. Thus, to be

mentally healthy a person must (page 3) "(i) be on good terms with himself; (ii) achieve good terms with others; (iii) possess sufficient skills to face all the varied situations of life".

The choice of a child guidance clinic as an instrument to promote community mental health is interesting, though we wonder how one man could act as professor, clinician and general coordinator of social, educational and psychological services, even with the projected assistance of trained Chinese staff. Projected extension of clinic services to include parents and teachers would surely demand an enormous institution requiring a broad interdisciplinary approach.

One of the main points of interest to Australians is the first-hand description of clinical problems posed by a Chinese community in a state of rapid change. A final encouraging feature is the outline of a programme for improving the mental health of teachers, an urgent task in both Hong Kong and Australia.

Disorders of the Blood: Diagnosis, Pathology, Treatment, Technique. By Sir Lionel E. H. Whitby, C.V.O., M.C., M.A., M.D. (Cantab.), F.R.C.P. (Lond.), D.P.H., and C. J. C. Britton, M.D. (New Zealand), D.P.H.; Eighth Edition; 1957. London: J. and A. Churchill, Limited. 9½" × 6", pp. 888, with 145 illustrations. Price: 75s. (English).

IN his preface, Britton writes: "It is with great sorrow that I am writing the preface of this edition alone, owing to the recent untimely death of Sir Lionel Whitby. At the time of his death the galley proofs of this edition had almost been completed." It is sad to realize that future editions of this great text-book will not have the benefit of the senior author's wisdom and experience.

Advances in hæmatology of recent years have been immense, especially with regard to the hæmorrhagic diseases and the importance of agencies which may have a toxic effect on the bone marrow. The chapters on "The Hæmolytic Anæmias" and on "The Purpuric and Hæmorrhagic Diseases" and many smaller sections have been rewritten. The account of hæmorrhagic diseases is adequate, yet admirably lucid, as is also the account of blood coagulation, included in the section on "The Principles and Practice of Hæmatological Diagnosis".

The book has been kept within bounds by the use of slightly smaller print, which seems rather a pity. Many will miss the clear, pleasant, familiar type of earlier editions. Is it necessary to keep the cost down to half that of a corresponding American publication? Chapter IX is still headed "Idiopathic Hypochromic Anæmia"—surely an anachronism and a misnomer, especially as the opening sentence reads: "Idiopathic hypochromic anæmia and chlorosis are classical examples of an anæmia due essentially to iron deficiency." There are a few surprising misprints and one of the plates is upside down. But these and other faults are minor ones; the new edition of "Whitby and Britton" keeps its place as the best British text-book of hæmatology.

The Clinical Application of Antibiotics: Volume III, Chloramphenicol and the Tetracyclines. By M. E. Florey, M.D.; 1957. London, New York and Toronto: Oxford University Press. 9½" × 6", pp. 413, with illustrations. Price: 12ls.

THIS important contribution contains a well-documented accumulation of basic information on the broad-spectrum antibiotics, chloramphenicol and the tetracyclines. Lady Florey has been most painstaking in her search of the literature, and has included many European references in addition to the considerable detail from American sources. The bibliography covers 44 pages.

Whilst the value of chloramphenicol in *Hæmophilus influenzæ* infections, particularly meningitis, and in Rickettsial infections has been emphasized, its important place in the treatment of penicillin-resistant staphylococcal infection receives scanty mention. The author accepts the view that chloramphenicol may be associated with the subsequent development of blood dyscrasia, but the incidence of fatal blood disorders in patients receiving the drug must be very low. Lady Florey points out that the drug should not be used unless there is bacteriological evidence and clinical experience to show that no other drug will be as effective in controlling the infection to be treated. In general, the use of chloramphenicol should be limited to serious conditions such as influenzal meningitis, typhoid fever, certain urinary tract conditions infected with organisms resistant to other antibiotics, and typhus if the response to the tetracyclines is delayed.

Two-thirds of the contents of the book are devoted to an excellent survey of the discovery and laboratory and clinical details of chlortetracycline, oxytetracycline and tetracycline itself. The complications of therapy are included, and the well-known symptoms of gastro-intestinal disturbance with stomatitis, diarrhœa and *pruritus ani* are enumerated, though not fully explained. In spite of the voluminous literature available on the use and complications of broad-spectrum antibiotics, the full explanation as to why some patients react as they do is still not forthcoming. Some of the gastro-intestinal disturbances could be due to impurities, others to a diminution in the normal flora of the gut with consequent reduction in synthesis of vitamin *B* and an overgrowing of the normally present Monilia. The use of vitamins and the anti-monilial drugs ("Nystatin") does not eliminate these effects. In fact, one worker states that excess vitamins may stimulate the growth of Monilia. The greatest danger is the rare complication of an intestinal infection ("superinfection") with a resistant *Staphylococcus aureus* producing pseudo-membranous ulcerative colitis. The question of sensitivity or allergy of the patient is mentioned. There is reasonable evidence that toxic effects are reduced, though not eliminated, by the use of the refined tetracyclines.

This is an excellent reference book for those wishing to delve deeper than the mail box and its blotter type of instruction in the use of the broad-spectrum antibiotics. However, events in this field move so rapidly that this book, having been published in 1957 with the latest reference to literature published in June, 1956, may not cover the immediate antibiotic therapy for a particular patient.

The Lower Urinary Tract in Childhood: Some Correlated Clinical and Roentgenologic Observations. By Sven Roland Kjellberg, Nils Olof Ericsson and Ulf Rudhe; 1957. Chicago: The Year Book Publishers, Inc. 10" × 7", pp. 310, with 265 illustrations. Price: $18.00.

THE study of the anatomy, physiology and pathology of the urinary tract in children has undergone a new and sustained stimulus since the introduction of micturition cysto-urethrography as a routine investigational procedure. The lower part of the urinary tract can be demonstrated in infants and children in almost all instances by proper management and handling of the patient. Micturition cysto-urethrography is probably the most informative and reliable single investigation of this part of the urinary tract. In many young patients the upper part of the urinary tract also can be demonstrated during the investigation by vesico-ureteral reflux.

This book reveals in detail the cysto-urethrographic appearances of the bladder and urethra in health and disease based on the investigation of some 1461 subjects; it points out clearly the normal contours and the abnormalities; it gives generous appraisement of the work of others in this field, but states the opinions of the authors where they are at variance with the literature. Ureteroceles, vesico-ureteral reflux, urethral valves, bladder neck obstruction, neurogenic bladders, urethral strictures, polypi and diverticula, enuresis radiography in intersex problems and many other aspects of children's urology are discussed. The illustrations are so free that the book could almost be stated to be an atlas; the quality of the illustrations is superb; the technique of obtaining simultaneous X-ray pictures in two planes is ingenious and important. A barium suspension in a watery medium is preferred by these authors to iodide solutions, on the grounds that it is non-irritant to the urethra and improves the density and quality of certain radiographs.

In a book of this type, which is a record of the author's own experiences and observations, many contentious points are aired. The chapter on ectopic ureter and ureterocele is clear and concise. The term ectopic is used in a restricted sense, and may be confusing unless the authors' definition is strictly observed throughout the chapter. In the discussion of the neurogenic bladder, many forms of conservative management are described with the varying degrees of success attending each. Judged by current writings since this book was written, more radical forms of urinary diversion operations such as the ileal conduit are now in vogue. In the references to both neurogenic disturbances of the urinary tract and urinary tract infection associated with vesico-ureteral reflux, emphasis is placed on infection as the cause of the reflux. Some observers would incline to the opposite view—that infection is caused by reflux which, in turn, is caused by some congenital defect in the ureter. Stenosis of the vesical outlet is considered to be rare in children, a conclusion with which we fully

agree. It is to be hoped that this chapter will dissuade surgeons from excising the bladder neck in children except in very rare instances. However, micturition cysto-urethrography is not the whole answer in diagnosis of the condition. More knowledge has yet to be gained before the diagnosis can be made with certainty in many instances.

The outstanding contribution is the excellent chapter on valves of the urethra. It is freely illustrated showing superbly the valve formations in the urethra. The authors' record of surgical treatment of 40 patients is exceptional and must be the envy of most urologists. Only two died, and 37 were totally relieved of obstruction as disclosed by pre-operative and post-operative radiographs. The choice of operation in the majority of their cases was transurethral resection. On rather unconvincing evidence, coincident bladder-neck resection was advised in the treatment of some children.

These authors are to be congratulated on their efforts to record their findings in this clear and excellent fashion. The book consists of 298 pages, includes a very full list of references and an index, and is beautifully illustrated throughout. The Year Book Publishers, who have reproduced in this volume their usual excellent standard, are to be congratulated. All urologists would find it stimulating and interesting. It is an essential addition to the pædiatric surgeon's libary. For the radiologist, it will take its place amongst his reference works.

Practical Electrocardiography. By Henry J. L. Marriott, M.D.; Second Edition; 1957. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6", pp. 248, with 158 illustrations. Price: 55s.

THIS text has been written for senior medical students and general physicians, who require a concise primer of electrocardiography without detailed consideration of the theoretical aspects of the subject.

Interpretation of electrocardiograms has many pitfalls, and the author emphasizes that the "Profession and laity alike are inclined to lay too much stress on mechanical devices in diagnosis. The electrocardiogram, which is far from infallible, is no exception. . . . It cannot be too emphatically urged that the electrocardiogram should always be read in the clearest possible light of clinical observation".

As in the first edition, much of the book is devoted to the elucidation of abnormalities of rhythm and conduction, but the sections on abnormalities of the ventricular complex have been enlarged.

Pleasing features of the book are the excellent illustrations, 50 more in this edition, and an index of illustrations. Clear-cut tabulated statements of diagnostic criteria are valuable to the student. A number of review tracings are included for the student to test his grasp of the subject. The value of this book is indicated by the need for two reprintings and a second edition within three years of its publication.

Connective Tissue. Edited under the direction of R. E. Tunbridge, Madeline Keech, J. F. Delafresnaye, and G. C. Wood; 1957. Oxford: Blackwell Scientific Publications. 8¾" × 5½", pp. 384, with illustrations. Price: 42s. (English).

THE Council for International Organizations of Medical Sciences, a body set up by UNESCO and WHO, organized a symposium on "Connective Tissue", which was held in London in July, 1956. The original papers presented and précis of the discussions have now been published in book form. The 30 participants were selected with great care from the leading workers in the field, and from many countries, including Russia and Hungary.

Connective tissue is being studied in very great detail at present, and several symposia have been held during the past few years. This one is by far the most detailed and authoritative. There are 26 papers on all aspects of the subject, together with an "Introduction" by W. T. Astbury and a "Summing Up" by the same scientist, who has done much work on the structure of connective tissue proteins.

It is not possible here to go into detail of the ground covered, but the cover is complete, dealing as it does with connective tissue fibres, their nature, origin, importance and relation to each other, the polysaccharides of connective tissue, other protein connective tissue and mast cells. This is the most complete account available of the nature and structure of connective tissue, and is recommended to anyone desiring information on the subject. It is not always easy reading.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Medicine: Essentials for Practitioners and Students", by G. E. Beaumont, M.A., D.M., F.R.C.P., D.P.H.; Seventh Edition; 1958. London: J. and A. Churchill, Limited. 9" × 5½", pp. 868, with 67 illustrations. Price: 45s. (English).

Thoroughly revised since the sixth edition was published in 1953.

"A Woman Doctor Looks at Love and Life", by Dr. Marion Hilliard; 1958. London: Macmillan and Company, Limited. 7½" × 5", pp. 192. Price: 10s. 9d. (English).

The author, former chief of obstetrics and gynæcology at Women's College Hospital, Toronto, has just retired after 25 years of active practice.

"An International Nomenclature of Yaws Lesions", by C. J. Hackett, M.D., F.R.C.P.; 1957. Geneva: World Health Organization. 9½" × 6", pp. 104, with 34 illustrations. Price: £1 (English).

The text has been "drawn from the opinions of many past and present authorities on yaws from many countries".

"Chromatographic Techniques: Clinical and Biochemical Applications", edited by Ivor Smith, B.Sc., Ph.D., F.R.I.C.; 1958. London: William Heinemann (Medical Books), Limited. 8½" × 5", pp. 324, with illustrations. Price: 45s. (English).

Is mainly concerned with the editor's own apparatus and techniques.

"High Arterial Pressure", by F. H. Smirk, M.D., F.R.C.P., F.R.A.C.P.; 1957. Oxford: Blackwell Scientific Publications. 9½" × 6", pp. 810, with many illustrations. Price 75s. (English).

Professor Smirk's extensive experience and original work in this field are well known.

"British Pharmacopœia 1958", published under the direction of the General Medical Council; 1958. London: The Pharmaceutical Press, published for the General Medical Council. 9" × 5½", pp. 140, with illustrations. Price: £3 3s. (English).

This "supersedes all previous editions, including the British Pharmacopœia 1953, and the Addendum 1955 thereto".

"Marriage and Personality: A Psychiatric Study of Inter-Personal Reactions for Student and Layman", by Ainslie Meares, M.B., B.S., B.Agr.Sc., D.P.M.; 1958. Springfield, Illinois: Charles C. Thomas, Publisher. 8" × 5½", pp. 158. Price: 30s. (English).

Intended to bring technical knowledge within the reach of the lay public.

"Child Psychiatry", by Leo Kanner, M.D., with Prefaces by John C. Whitehorn, M.D., Adolf Meyer, M.D., LL.D., and Edwards A. Park, M.D.; Third Edition; 1957. Oxford: Blackwell Scientific Publications. 10" × 6", pp. 804, with illustrations. Price: 63s. (English).

The author is Professor of Child Psychiatry in The Johns Hopkins University.

"Antisera, Toxoids, Vaccines and Tuberculins in Prophylaxis and Treatment", by H. J. Parish, M.D., F.R.C.P.E., D.P.H.; Fourth Edition; 1958. Edinburgh and London: E. and S. Livingstone Limited. 8½" × 5", pp. 266, with many illustrations. Price: 30s. (English).

The author is Clinical Research Director of the Wellcome Foundation Ltd.

"Practical Clinical Chemistry: A Guide for Technicians", by Alma Hiller, Ph.D.; 1958. Second Edition, 1958. Springfield, Illinois: Charles C. Thomas, Publishers. 9" × 5", pp. 285. Price: 50s. (English).

Written mainly for technicians, this book describes procedures "chosen as representative analyses which are most frequently performed in a hospital laboratory of clinical chemistry".

The Medical Journal of Australia

SATURDAY, MAY 17, 1958.

THE JOHN CURTIN SCHOOL OF MEDICAL RESEARCH.

TEN years ago the Australian National University was little more than an idea. In a speech delivered at the University of Melbourne in April, 1948, which we quoted in a leading article[1] at the time, Professor (now Sir) Douglas Copland spoke of the basic plans then being discussed for the National University. His appointment as Vice-Chancellor of the new university had not long been made. He described the plans as "necessarily tentative", and referred to the difficulties of obtaining men and materials to carry out the project despite the fact that the Commonwealth Government had provided sufficient funds by statute to support the university "on a sound basis". Looking back on this statement now we find it hard to be sure whether the men and materials in short supply were those needed to raise bricks and mortar into buildings or those who would fashion ideas and equipment into a first-class university—presumably it was the former. Certainly advice had already been sought from such men as Sir Howard Florey and Professor Marcus Oliphant; and Professor Copland said that it was hoped to develop "one of the leading centres in the world of research in nuclear physics" under the guidance of Professor Oliphant, and that it might be possible for research work in medicine to be commenced under Sir Howard Florey's guidance before the medical laboratory was complete. At the time we referred to the rather vague phrase "under the guidance of" and commented:

> We should like to read into the words a declaration that these two leaders in research were coming to Australia to take charge of work at Canberra, to bring their alert minds to the solution of world problems while they are living among us and to stimulate Australian workers. We fear that this is not so.

The intervening years have seen notable developments, and the Australian National University is now well established in Canberra. Despite difficulties, men and materials have been made available to raise buildings, and first-class men with first-class equipment are building the more intangible but infinitely more valuable structure that will, we hope, prove to be an outstanding research centre. Our gloomy prognostications about leadership have not been wholly justified. Professor Marcus Oliphant is now the Director of the Research School of Physical Sciences, which is housed in a suitable and permanent building at Canberra, and undoubtedly, although the subject is well outside our legitimate sphere of comment, important work is being done there. The Research Schools of Social Sciences and of Pacific Studies are developing under the leadership of Sir Keith Hancock and Dr. J. W. Davidson respectively. The permanent building of the fourth school, the John Curtin School of Medical Research, which is naturally the one of greatest interest to us, has now been completed and was officially opened by Sir Howard Florey on March 27, 1958. The occasion is reported elsewhere in this issue.

Undoubtedly the genesis and the development of the John Curtin School of Medical Research owe a great deal to Sir Howard Florey, and it was fitting that he should open the new building. Whether or not he is likely ever to be its director we are not in a position to know, and he gave no hint on the matter in his speech. Perhaps we may still hope that this will be so. However, be that as it may, the John Curtin School has a first-rate team of leaders. The heads of four of the five departments have held their appointments since early in the school's development. These are A. H. Ennor (Professor of Biochemistry and Dean of the School), Adrien Albert (Professor of Medical Chemistry), F. J. Fenner (Professor of Microbiology) and J. C. Eccles (Profesor of Physiology). The fifth, F. C. Courtice, who has been Director of the Kanematsu Memorial Institute of Pathology at Sydney Hospital, has just been appointed Professor of Experimental Pathology. Each of these men is a graduate of an Australian university, and each already stands high in his own field in any company. With each is associated a keen and able group of whom more will undoubtedly be heard. The first four of the departments were initially housed away from Canberra. The Department of Biochemistry was formed in the Commonwealth Serum Laboratories in Melbourne and the Department of Microbiology in the Walter and Eliza Hall Institute of Medical Research, also in Melbourne. The Department of Physiology began in the University of Otago, New Zealand, and the Department of Medical Chemistry in the laboratory of the Wellcome Foundation, London. Temporary quarters were provided in Canberra in 1951, and the Departments of Biochemistry, Microbiology and Physiology moved there in 1952. In 1953 the Department of Experimental Pathology was formed by three research workers from Sir Howard Florey's department at Oxford. All five departments are now suitably housed in the John Curtin School.

Essentially a research centre, with no undergraduate teaching role, the John Curtin School of Medical Research will have its own place in the academic medical world in Australia; and, if we may judge by Sir Howard Florey's remarks, it is not intended to develop on an "ivory tower" basis, far removed from clinical medicine, but will have its own part to play in stimulating clinical research. The other five Australian medical schools, all of which are actively concerned with the teaching of undergraduates as well as with research and post-graduate teaching, are just now feeling and responding in their several ways to the strong currents of modern thought on medical education and the development of medical science. Two others, still in the planning stage, have the opportunity of a fine start if there is no bungling. It is a great day for medicine everywhere, and not least in Australia. The tide is ready to be taken at the flood. The John Curtin School of Medical Research, integrated with the medical schools of the other Australian universities, could provide the additional stimulus that would lift Australian medicine to great heights.

[1] M. J. AUSTRALIA, 1948, 2: 43 (July 10).

Current Comment.

THE WHEEL AND HEALTH.

THERE has been a trend through the ages towards a steadily increasing replacement of personal effort by mechanical devices, and this trend has been much accelerated in the past 50 years in the wealthier countries. It has been said that man's first mistake was the wheel. R. T. Smith,[1] in discussing the causation and treatment of chronic fibrositis or, better, muscular rheumatism, writes:

> Our idolatrous worship of the wheel is being communicated to the younger generation to such an extent that the former U.S. Director of Physical Fitness under the Civilian Defense Programme in World War II felt impelled to report that "57·9% of American youngsters failed in one or more of six basic strength and flexibility tests, whereas only 8·7% of the European children failed them" (Kraus-Weber tests).

The European children were examined in Austria, Switzerland and Italy. W. Raab[2] discussing the same observations, in an article on "Loafer's Heart", puts the results down to general lack of exercise, such as walking, running and carrying, affecting the muscular fitness of the motorized and TV-sitting young generation of the U.S.A. Both authors point out that lack of use of muscles leads to atrophy, often over a long period. This is well seen after rest in bed for several weeks. Raab states that there is one unique muscle—the myocardium—which cannot be put safely to permanent relative rest and thus made incapable of coping with acute demands to which even the most civilized physical loafer is occasionally exposed. Raab quotes some most impressive data obtained by J. N. Morris on British workers. These suggest that there is a reversed proportion between the degree of physical activity on the one hand and mortality from coronary heart disease on the other. Hairdressers and office clerks give the highest number of cardiac deaths, whereas heavy agricultural and industrial labourers appear less affected. In Denmark and Holland, where the bicycle and not the automobile is the typical national means of transportation, the cardiac death rate is considerably lower than in the United States, despite an almost equally high fat consumption.

Raab goes on to state that much is known about relations between muscular exercise and neurovegetative action on the heart. Myocardial oxidative metabolism and energetic efficiency are controlled by the opposing actions of the adreno-sympathetic substances (adrenaline, arterenol) and vagal acetylcholine. Sympathetic preponderance, whether actual or relative, is dangerous to myocardial functional and even structural efficiency. Cholinergic vagal preponderance improves the oxygen economy of the heart muscle and increases efficiency. Systematically trained athletes show highly developed vagal tone, as demonstrated amongst other things by bradycardia at rest and small stroke volume at rest with great augmentation during exercise. Where there is cardiac degeneration these factors are reversed. Three weeks of complete bed rest (in healthy volunteers) produces a marked and prolonged preponderance of adrenergic cardiac manifestations. The vascular supply of the heart is also subject to alterations connected with physical activity and inactivity. The capillary network of athletes' hearts is unusually richly developed. The relation of physical inactivity to coronary atherosclerosis is not yet known.

R. T. Smith believes and brings forward much evidence to indicate that chronic fibrositis is caused by a decrease in muscle strength, bulk and tone designated as atrophy. It may be caused by disuse, decreased use or prolonged muscular strain. Decreased use of muscles due to change in habits will not produce symptoms for a year or two, but they appear sooner or later if the decrease is sufficient. When atrophy develops, the individual muscle fibres (and therefore the whole muscle) becomes thin and elongated, so that there is actual slackness of the muscle and lack of tone. There is then a slightly prolonged stimulus-reaction time, and the muscle must contract through a greater distance; hence there is earlier fatigue. As the condition develops, there is spasm of the muscle or stiffness known as "jelling". It is seen that fibrous tissue takes no part in the muscle changes; so the name fibrositis is a misnomer, and muscular rheumatism is a better term.

Smith believes that regulated exercises for the disused muscles with salicylates and heat will cure chronic fibrositis in a few weeks. An elastic roller bandage, firmly applied approximately four finger-breadths above the patella or above the wrist (with gauze or sponge rubber padding to prevent undue pressure over the arteries of the forearm), will take up the slack in the muscle of the leg or forearm respectively and give great relief and increased use of the muscles with less fatigue. "Chronic fibrositis can be cured in a relatively short period of time if the treatment is individualized and cooperation of the patient is secured." This involves the development of exercises which will contract the affected muscles as completely as possible by exercising against resistance. The exercises are carried out for a few moments every waking hour. Smith points out that it is difficult to get this cooperation in a community which believes in the heresy—"don't walk if you can ride; don't stand if you can sit; don't sit if you can recline; and if you get the urge to exercise, lie down till it passes off". But the results are worth the effort.

While it would undoubtedly be better for the general health if everybody used their muscles—cardiac and skeletal—to a much greater extent than is usual in the wealthier countries, it is doubtful whether this will happen on any large scale, and modern medicine must develop some other means of compensating for the disturbed central nervous and peripheral neurovegetative equilibrium if we want to escape further degeneration. Meanwhile common sense says: "Exercise."

[1] *J. Am. Geriatrics Soc.*, 1958, 6: 147 (February).
[2] *Arch. Int. Med.*, 1958, 101: 194 (February).

AURAL SURGERY.

ON another page we publish an article by A. B. K. Watkins in which he gives a brief account of recent developments in the surgical treatment of deafness. This is a subject in which spectacular advances have been made during the past decade, and which is still in a state of ferment. In an article published recently S. Rosen,[1] who pioneered the operation of stapes mobilization, has reviewed the developments in this particular corner of aural surgery during the five years since he first introduced the operation, and his paper makes interesting reading in the light of Watkins's account. In his early attempts to mobilize the stapes Rosen found that continued pressure on the stapedial neck often caused the crura to fracture before the foot-plate could be mobilized, so he has evolved a more direct method of going to the foot-plate itself in order to mobilize it. In contrast to the simple myringotomy performed in earlier operations, he now removes sufficient of the overhanging bone of the postero-superior meatal wall to expose the oval and round window areas clearly, so that the stapes can be manipulated with precision. A sharp-pointed explorer is then gently wedged into the area between the bony rim of the oval window and the periphery of the foot-plate itself, usually at the inferior margin. The point of the explorer is moved ever so slightly in and out as if prying loose a lid. When successful, the operator suddenly see the entire stapes moving. Improvement of hearing can be obtained whether or not the crura have been fractured previously. When the fixation of the foot-plate is extremely rigid, owing to the extent of the sclerosing process, both the indirect method of pressure on the stapedial neck and the direct method may fail to produce mobilization. In such cases an attempt may be made to create a window through the rigid

[1] *Arch. Otolaryng.*, February, 1958.

footplate itself, using a sharp-pointed explorer; sometimes an explorer with a hooked tip is used, the tip being used to punch out a small piece of bone on withdrawal. Most often this puncture is made in the postero-inferior quadrant of the foot-plate as close to the junction of the posterior crus as possible.

Rosen's paper illustrates the extreme delicacy of touch necessary in modern aural surgery. Its development has been made possible only by the development of dissecting microscopes which enable the operator to work under high magnification and good illumination. Even so, Rosen emphasizes that much practice in the post-mortem room and on other materials is essential to develop a precise tactile appreciation of these procedures; and to the same end he operates without rubber gloves. A further illustration of the delicacy of technique required may be seen in the paper by A. L. F. Saft published in our issue of May 3, 1958.

Good results are claimed for the operation of fenestration of the foot-plate. Slight vertigo may be experienced for a few days after this procedure, but in over 200 operations Rosen has not had a single instance of labyrinthitis or injury to the facial nerve. Hearing has been restored to normal or near normal even when the ossicular chain has been interrupted by fracture of the crura, and in some instances in which the incus was removed to give better access. Of 210 attempts at fenestration of the oval window, 136 resulted in the creation of a definite opening into the vestibule. In the remaining 74 patients there was no definite assurance that an opening had been made, although in some of these cases the hearing showed improvement. Patients with better hearing by bone conduction have better chances of recovery of useful hearing. The improvements gained have been maintained for periods up to 21 months so far, and only nine out of 77 patients so improved have relapsed to the levels of hearing present before operation. The procedure of fenestration of the foot-plate appears to have given a higher percentage of restoration of useful hearing than either the indirect or the direct stapes mobilization procedure.

WORLD MORTALITY RATES.

A CONTINUOUS DECREASE in mortality from infectious and parasitic diseases and from complications of pregnancy and childbirth, and an increase in mortality from cancer of the respiratory system and heart and liver diseases are some of the modern health-trends confirmed by a recently published statistical report of the World Health Organization.[1]

In some of the 28 countries and territories covered by the report, mortality from complications of pregnancy and childbirth dropped almost 50% between 1950 and 1956. Among the countries reporting rates for 1956, Denmark had the lowest (48·2 per 100,000 live births) and Ceylon the highest (376·5); in Israel (Jewish population) the rate was 48·4, in Scotland 53·5, in France 55·3, in Australia 56·1, in England and Wales 57, in Northern Ireland 57·6, in Canada 61·7, in the Netherlands 67, in Ireland 85·6, in Finland 111·4, and in Austria 121·7. In the following countries, the latest rates given are for 1955: New Zealand 44·1, U.S.A. 47, Sweden 49·4, Norway 67·7, Belgium 80·4, Switzerland 104·3.

The striking decline in mortality from infectious and parasitic disease is set out in a table including all countries reporting rates from 1950 to 1956 (rates per 100,000 population). In practically every case the decline is a steady one, and it will be sufficient to quote the figures for 1950 and 1956 respectively for each country: Canada, 36·9, 14·6; Ceylon, 180·5, 91·3; Israel, 55·8, 17·7; Japan, 201·9, 69·0; Finland, 118, 48·4; France, 75·2, 48·9; Ireland, 91·2, 31·2; Netherlands, 30·7, 12·2; England and Wales, 47·5, 18·5; Scotland, 64·5, 20·7; Northern Ireland, 61·3, 20·7; Australia, 32·0, 15·3.

[1] *Epidemiol. & Vital Statist. Rep.*, Vol. II, No. 3, 1958.

Of the "modern" diseases, cancer of the respiratory system is the cause of death that is most rapidly growing in importance in the majority of highly developed countries. The steady increase in mortality from this cause is set out in a table including all countries reporting rates from 1950 to 1955 or 1956 (rates per 100,000 population). Again we quote only the figures for 1950 and 1955 or 1956 respectively for each country: Canada, 10·8, 14·6; U.S.A., 14·3, 18·5; Israel, 8·0, 9·2; Japan, 3·3, 4·7; France, 15·4, 19·3; Ireland, 10·4, 18·4; Netherlands, 14·9, 20·0; England and Wales, 31·2, 43·5; Scotland, 30·3, 37·9; Northern Ireland, 15·2, 26·6; Australia, 11·2, 14·5; New Zealand, 12·9, 18·2.

RAUWOLFIA IN THE TREATMENT OF MALNUTRITION.

PATIENTS suffering from various chronic diseases frequently show evidence of malnutrition and underweight, and the treatment of this presents many difficulties. Diets of high calorie and high vitamin content given with the appropriate treatment of the underlying disorder only occasionally help the malnutrition. L. G. Dinkin[1] has tried a variety of different additives such as antibiotics, large doses of vitamins, hormones and so on, but had little success until rauwolfia was given. It was an early observation when rauwolfia was introduced to western medicine that after administration of the drug the patients frequently put on weight and took their food better. Dinkin studied 42 patients who had shown evidence of malnutrition for from two months to three years. Either whole root preparations of rauwolfia or the alkaloid reserpine were given with a diet of high calorie and high vitamin content. Of the 42 patients 35 gained weight during the period of treatment. Slightly greater gains were observed with reserpine. Parallel with the increased weight there was an improvement in the patient's general condition, although most of the patients had incurable diseases. The patients consumed larger quantities of food while under treatment. Very few side effects were noted, and these were unimportant. When the rauwolfia was discontinued there was usually an arrest of weight gain or a loss in weight. It is suggested that the effect is due to an improvement in the emotional adjustment.

INDUSTRIAL SAFETY HELMETS

THE Standards Association of Australia has issued for public critical review a draft Australian standard for industrial safety helmets (Doc. 381). The draft standard applies to industrial helmets for use in industries in which there is danger of injury to the head. Materials and design are covered in general terms, and a range of sizes is specified. There are tests for mechanical strength and inflammability, and, if required, electrical resistance. The draft is based largely on the relevant British standards 2095 and 2826, covering light-duty and heavy-duty types respectively. There has been considerable argument within the committee on the type to be standardized for Australian purposes, and Doc. 381 incorporates the majority decision that, in view of recent availability of improved materials, the heavy-duty test should be adopted. Comment on this aspect will be welcome. The question of plain versus ribbed shells is another controversial aspect on which wider comment would be of value to the committee.

Copies of Doc. 381 may be obtained from the Headquarters of the Association, Science House, Gloucester and Essex Streets, Sydney, and from branch offices in capital cities and at Newcastle. Comment is sought from those with experience in the manufacture, supply and use of industrial safety helmets and should be forwarded to the Association not later than June 2, 1958.

[1] *New York State J. Med.*, 1958, 85: 505 (February 15).

Abstracts from Medical Literature.

RADIOLOGY.

Radiological Changes After Mitral Commissurotomy.

J. G. McAFEE AND P. BIONDETTI (*Am. J. Roentgenol.*, August, 1957) report on the results of "follow-up" X-ray examination of 150 patients after mitral commissurotomy. They state that "B" lines, first described by Kerley, have been definitely established histologically as pulmonary interlobular septa. They appear as thin, parallel, horizontal, dense lines in the lung bases extending out to the pleural surfaces and are best seen just above the costophrenic angles, usually in the frontal projection, but occasionally in the oblique views. In mitral valvular disease, these abnormal lines are thought to be due to œdema and lymphatic dilatation in the interlobular septa, secondary to pulmonary hypertension. "B" lines were present in 33% of the patients in this series. The presence of "B" lines was not closely correlated with the level of pulmonary hypertension in those patients who were subjected to cardiac catheterization. When the lines were marked, fairly severe hypertension was invariably present, but a few more of the cases with the greatest pulmonary hypertension had no visible lines. When these lines were minimal in extent, they had no correlation with the level of pulmonary hypertension. There was, however, a fairly good correlation with the calculated pulmonary arteriolar resistance. Comparing patients with and without "B" lines, the clinical results following surgery were found to be identical. This was to be expected, since the clinical results were not materially affected by the severity of the pulmonary hypertension. The lines were observed in patients with mitral insufficiency as well as in those with mitral stenosis. Observation of the "B" lines before and after surgery proved valuable, because the disappearance of the lines a few weeks after surgery was often the first objective evidence of improvement. In 24 patients in whom the "B" lines promptly disappeared, the clinical results were invariably satisfactory. On the other hand, the clinical results were poor in six out of nine patients in whom the "B" lines persisted for several months after operation.

Pulmonary Arterio-Venous Fistula.

I. STEINBERG AND N. FINBY (*Am. J. Roentgenol.*, August, 1957) state that pulmonary arterio-venous fistulæ are hereditary hæmangiomatous malformations of the pulmonary vascular bed. Almost 40% of the reported cases also have hæmangiomata elsewhere in the body, indicating that a close relationship to familial telangiectasis exists. When the pulmonary arterio-venous fistulæ are large and do not have systemic arterial connexions, dyspnœa, cyanosis, clubbing of the digits and polycythæmia result. Hæmoptysis may occur when there is rupture of the thin pulmonary vascular wall. There is also a high incidence of cerebral symptoms due either to cerebral thrombosis secondary to polycythæmia or to brain abscess secondary to chronic pulmonary infection. The heart remains normal in size because pulmonary arterio-venous fistulæ do not cause increased pulmonary vascular resistance. In the authors' series of 13 cases the classic syndrome of cyanosis, clubbing of digits, polycythæmia and a vascular murmur heard over the pulmonary fistula was found only twice. One patient with dyspnœa, fatigue and familial hæmorrhagic telangiectases had bilateral pulmonary arterio-venous fistulæ. In five instances the patients were asymptomatic, in eight the pulmonary arterio-venous fistulæ were responsible for symptoms, and in three of these acute cerebral conditions (brain abscess, meningo-encephalitis and hemiplegia) were present. Vascular bruits were heard in 11 patients; all had abnormal chest skiagrams. The diagnosis of pulmonary arterio-venous fistula can often be made by conventional radiography, particularly if there is reason to suspect such a lesion. Pulmonary densities with hilar vascular connexions localized in lobes or segments of the lungs, and increased hilar vascularities, should arouse suspicion of an arterio-venous fistula. Change in size of the lesions after respiratory manœuvres and delineation of the afferent arterial and efferent venous connexions by laminagraphy conclusively establish the diagnosis, and are especially recommended for pre-operative evaluation. The treatment of pulmonary arterio-venous fistulæ is surgical excision, with conservation of as much lung tissue as possible in order to preserve pulmonary function, especially if multiple lesions are present. Surgery is usually curative. It is recommended even in the asymptomatic case since the risk of brain abscess, fatal hæmoptysis or development of cerebral thrombosis when polycythæmia occurs is far greater than the morbidity and mortality of lobectomy or segmental resection.

Whiplash Injuries of the Cervical Spine.

D. B. NAGLE (*Radiology*, December, 1957) reviews some of the less obvious radiographic evidences of cervical spine injuries resulting from the so-called whiplash mechanism. A true whiplash injury results from hyperflexion of the neck caused by a violent forward thrust of the head when the motion of the body has been suddenly slowed or stopped, as in a head-on collision, or a recoil hyperflexion produced by acceleration of the body from behind. The relatively large weight of the human head which is supported on a narrow column of bone, coupled with the extreme mobility of the cervical spine, provides the mechanical basis for this type of injury. The types of fractures most commonly encountered in whiplash injuries are those involving the lateral masses, posterior arches, transverse or spinous processes of the vertebræ, or chip fractures of the anterior margins of the centra. Odontoid process fractures are not unusual. Fractures of the compression type, commonly met with in other parts of the spine, are of less frequent occurrence in the cervical region. Ligamentous injury as a result of whiplash trauma occurs just as frequently as fracture, and its recognition is probably nearly as important. However, this type of injury is often overlooked. For an understanding of these injuries one must recall the normal mechanics of the cervical spine, and particularly the usual range of movements. Flexion and extension involve a gliding motion of each vertebral body upon the one beneath, as well as a rocking motion centred about a pivotal point located at the junction of the centrum and pedicles. The range of these movements is greatest in the mid-cervical spine, a fact that is reflected in the earlier appearance of degenerative arthritis in that area. Normally, with the neck in neutral position, the alignment is an uninterrupted curve, convex anteriorly, following the line of the posterior margins of the vertebral bodies. When injury from hyperflexion occurs, it begins as a tear of the capsular structures of the posterior joints, followed in turn by rupture of the interspinous ligaments, tears of the fibres of the *annulus fibrosus* and actual disk injury. With greater stress, subluxation and/or fracture occur. The first stage of hyperflexion injury without ligamentous damage produces muscle strain, which will result in muscle spasm, with straightening or even reversal of the normal curvature and limitation of all movements. There is, however, no focal disruption of the curve or of the alignment of the spinal elements. With a lesser degree of ligamentous injury, in which there is only partial avulsion of posterior joint capsular structures, the curve is altered and there is localized reduction of the normal range of intervertebral movement and straightening of the curve.

Glomus Jugulare Tumours of the Temporal Bone.

R. A. K. HARPER (*J. Fac. Radiologists*, July, 1957) discusses glomus tumours affecting the mastoid area and originating in the region of the jugular bulb within the temporal bone. The radiological features which are encountered in this type of tumour vary considerably according to the extent of the tumour, and they are described in relation to the gradual extension of the tumour. Sclerosis of the mastoid and petrous regions is sometimes the first sign, and at this stage simulates an old infected mastoid, but tomography will probably reveal a certain irregular erosion within the dense area. Mastoid infection may be secondary, due to obstruction of the drainage from the mastoid process; or a glomus tumour may arise coincidently in a patient with a past history of mastoid infection. In either case tomography is important to demonstrate the early stages of destruction of the petrous bone as the tumour spreads from the region of the jugular bulb. At a later stage the middle ear and labyrinth tend to be destroyed and large defects appear in the petrous bone. Until very late in the disease, the tegmen usually remains, although it may be elevated in the later stages. In advanced stages, the whole of the petrous area and much of the mastoid and squamous portions of the temporal bone may disappear. In the infero-superior position the jugular foramen may be seen to be enlarged in the early stages, and later to involve the carotid canal and to

extend into the apex of the petrous area. The tumour may spread medially to erode the articular process of the occipital bone, and anteriorly to invade the temporo-mandibular joint in its medial aspect. This involvement is best seen in the Towne position, or by tomography. As the tumour is highly vascular, the groove for the emissary vein of the mastoid process may be enlarged, and in advanced disease may be very large indeed. As the invasion of the occipital and petrous bones proceeds, it is common to see the vertebral canal in the first cervical vertebra much more clearly, and the foramen can often be observed to be larger than on the opposite side. Carcinoma of the middle ear also causes extensive destruction of the mastoid area, but the destruction tends to spread more laterally than medially, so that the apex of the petrous bone is not so widely destroyed, nor is the inferior destruction of the petrous bone to be seen, which is the usual finding in glomus tumours. Cholesteatoma may cause gross destruction of the mastoid area, but does not extend so far medially or inferiorly as does the glomus tumour, and the defect is usually well defined. A nasopharangeal tumour may invade the base of the skull and the inferior aspects of the petrous temporal bones, but will not produce the unilateral destruction characteristic of a glomus tumour. Only in the rare cases of bilateral glomus tumours is difficulty likely to arise, and then the presence of a large mass in the nasopharyngeal space will indicate the true diagnosis. Acoustic neuroma may cause difficulty in the differential diagnosis because of the extent of destruction of the petrous bone which may occur, but this is usually well defined and there is no inferior destruction or moth-eaten erosion of the remainder of the mastoid area such as is seen in an extensive glomus tumour.

Farmer's Lung.

R. C. FRANK (*Am. J. Roentgenol.*, February, 1958) reviews 27 cases of farmer's lung, outlining the variations in onset and course. Two fundamental reactions are considered to be involved in the disease process. There is an initial pulmonary response to inhalation of organic dusts involving a granulomatous reaction with associated interstitial fibrosis. Later sensitization to such dusts is believed to occur, and a progressive pulmonary fibrosis develops as a result of repeated exposures. Some permanent pulmonary disability may follow the initial attack and, once sensitization has occurred, increasingly severe disability is certain to take place after repeated exposures. The dusts involved are most often due to mouldy hay, grain or silage, and the heavy fungal spore content of such dusts is believed to be largely responsible for the disease, although rarely the syndrome is due to vegetable dusts which are not significantly mouldy. The pulmonary reaction to the fungal spores is due to their action as inhaled foreign matter and the disease is not believed to represent a pulmonary infection or mycosis. For the majority of patients developing the disease, even if only a single attack, a change of occupation should be strongly recommended. The author suggests that this disease should be regarded as a form of pneumonoconiosis due to organic dusts. The characteristic radiographic changes in the acute stage consist of fine to coarse granular and nodular mottling, scattered quite diffusely throughout both lung fields, although it is less prominent in the apices and peripheral bases. Changes on the chest skiagram may be very slight at the beginning of the first attack, and in some cases these changes developed long after exposure to dust and the first appearance of symptoms. In general, the appearance of radiographic changes coincides with the greatest severity of symptoms. In none of the cases presented was significant evidence of hilar or mediastinal lymphadenopathy found. The presence of such lymph node enlargement would therefore weigh heavily against a diagnosis of farmer's lung. Although the individual parenchymal lesions varied considerably in size and frequently appeared to be quite irregular and poorly circumscribed, a characteristic frequently noted was a very fine "sand-storm" appearance consisting of uniformly distributed fine granular densities involving various areas of the lung. These individual lesions are about a millimetre or less in diameter, as seen on the skiagram, and are discrete. Clearing takes place gradually, and although the X-ray appearance of the chest may return to normal after an acute attack, mild emphysema and scattered fibrotic strands are not infrequent sequelæ. In the subacute stage of the disease, when there is continued exposure to the causative agent, coarsening of the truncal markings may be observed, suggesting an active interstitial process which may lead to fibrosis, pulmonary hypertension and eventually *cor pulmonale*. The final stage consists of severe pulmonary fibrosis and emphysema with accompanying right heart strain.

RADIOTHERAPY.

Cancer of the Tongue.

S. CADE AND E. S. LEE (*Brit. J. Surg.*, March, 1957) state that since 1925, 653 patients with carcinoma of the tongue have been seen at the Westminster Hospital. The sex ratio has fallen from 10 males to one female in 1925, to two males to one female in recent years. A general decrease of deaths from carcinoma of the tongue is due entirely to decrease in the disease in males, probably due to the decrease of syphilis, severe oral sepsis and leukoplakia. Multiple primary lesions occurred in 4·1% of these patients, and all but eight lesions were squamous cell carcinomata. Most of the lesions were in the anterior two-thirds of the tongue, and were treated with radium implants. The optimal dose rate seemed to be 30*r* to 60*r* per hour to a total of 7000*r*. The operative mortality (573 implants in 507 patients) was 2·6%. The authors avoid large volume implants and combinations of interstitial and external radiation therapy. In posterior tongue lesions they consider teletherapy using gamma sources or two-million-volt sources the treatment of choice. Radical surgery for primary lesions was considered to be too mutilating and was usually limited to recurrences and radio-resistant lesions. Involved cervical lymph nodes were present in 57·5% of all patients when first seen. Full block dissection was performed whenever possible (117 cases), after the primary lesion was controlled. Neither partial (sub-mandibular) nor prophylactic neck dissections were undertaken. Teletherapy or radium implantation in the neck was used whenever surgery was not possible. The over-all five-year survival rate was 27%. Five-year survival for those with involvement of the anterior two-thirds of the tongue was 35%, whereas only 11% of those with posterior third involvement survived for five years. Of patients in whom enlarged lymph nodes were present when they were first seen, 14% lived five years, whereas 51% of those without enlarged lymph nodes when first seen lived five years. Other unfavourable factors were a large primary lesion (over two centimetres in size) and a primary lesion which crossed the mid-line or involved adjacent structures. Survival figures for females were better than for males, due to earlier diagnosis in the women. The degree of cellular differentiation did not seem to have any prognostic value.

Leukæmia After Radioactive Iodine Therapy.

S. C. WERNER AND E. H. QUIMBY (*J.A.M.A.*, November 23, 1957) report the case of a 28-year-old woman who developed acute leukæmia 18 months after being treated for hyperthyroidism with I^{131}. The treatment dose was given in divided amounts to avoid the consequences of possible undue sensitivity to radiation effect, and totalled 2·1 millicuries. This would have resulted in the administration of between one and three rads of general body exposure, and between two and six rads to the blood. The authors quote a very similar case, reported in Britain, in which a patient developed acute leukæmia 18 to 24 months after the administration of a total of 7·1 millicuries of I^{131}. They point out that the spontaneous incidence of acute leukæmia in the population as a whole is estimated to be about one per 20,000 persons per year, and that it has been calculated that this rate would be doubled by general body exposure of between 30 and 50 rads. Such dosage levels are in fact employed in the treatment of thyroid cancer, when a calculated risk is justified, but in the treatment of hyperthyroidism the dosage usually employed is well below this level. They state that at a recent conference the histories of 13,000 patients, from several clinics, who had been followed for from one to 10 years after I^{131} therapy for hyperthyroidism, were available for review. Four of these patients had developed acute leukæmia after treatment; this corresponds to a rate of one per 16,000 patients per year. This rate approximates sufficiently closely to that in the general population to justify the conclusion that the occurrence of such cases as have been reported is due to coincidence. However, the authors agree that all occurrences of leukæmia in patients who have received I^{131} therapy should be recorded in detail in order to establish whether there is or is not any connexion.

Special Article.

CARCINOMA OF THE UTERUS.

THE following article, which relates to the results of treatment of carcinoma of the uterus, consists of a summarized report (slightly adapted) compiled by Hans Ludvig Kottmeier, editor of the "Annual Report on the Results of Treatment in Carcinoma of the Uterus, Radiumhemmet, Stockholm", and an addendum showing figures from the Royal Prince Alfred Hospital, Sydney. The information has been made available by Sir Herbert Schlink, on behalf of the staff of King George V Memorial Hospital, Sydney.

TABLE I.

Result of Treatment.

Result.	Total.	Latest Five Years.
Alive with no evidence of the disease	35,811 (36·5%)	14,599 (41·5%)
Alive with carcinoma present	1,492 (1·5%)	561 (1·6%)
Died of carcinoma	56,081 (57·1%)	18,467 (52·4%)
Lost sight of	2,218 (2·2%)	531 (1·5%)
Died from intercurrent disease	2,616 (2·7%)	1,048 (3·0%)
Total	98,218	35,206

Summarized Report on Results Obtained in 1949 and Previous Years (Collated in 1956).

This summarized report is made up in accordance with "Comments" in Volume 10 of the "Annual Report on the Results of Treatment in Carcinoma of the Uterus", although we consider that the existing division of cases of carcinoma

TABLE II.

Distribution by Stages.

Stage.	Total.	Latest Five Years.
Stage I	17,292 (17·6%)	6,945 (19·7%)
Stage II	36,557 (37·2%)	13,386 (38·0%)
Stage III	34,773 (35·4%)	12,372 (35·2%)
Stage IV	9,589 (9·8%)	2,503 (7·1%)
Unclassified	7	0
Total	98,218	35,206

of the corpus can be discussed. The Report includes statements from 78 institutions in 20 different countries. Statements on carcinoma of the cervix have been submitted by all collaborators and on carcinoma of the corpus by 27 collaborators.

TABLE III.

Relative Five-Year Recovery Rate Calculated for Each of the Four Stages, Including All Years Reported On.

Stage.	Number of Patients Treated.	Alive with No Evidence of the Disease.	Relative Apparent Recovery Rate (%).
Stage I	17,292	10,870	62·9
Stage II	36,557	15,843	43·3
Stage III	34,773	8,475	24·4
Stage IV	9,589	623	6·5
Unclassified	7	0	0·0
Total	98,218	35,811	36·5

Carcinoma of the Cervix.

The submitted material relating to carcinoma of the cervix furnishes information concerning 98,218 treated cases.

The five-year results obtained in the treatment of carcinoma of the cervix and the distribution by stages of the cases treated are shown in tabular statements (Tables I, II, III and IV).

The over-all relative apparent recovery rate of 36·5% in this Report is the highest yet presented in the "Annual Reports", the comparative rates being: Volume 2, 26·3%; Volume 3, 27·6%; Volume 4, 26·7%; Volume 5, 30·9%; Volume 6, 31·8%; Volume 7, 32·6%; Volume 8, 34·1%; Volume 9, 34·7%; Volume 10, 35·3%.

TABLE IV.

Relative Five-Year Recovery Rate Calculated for the Latest Five Years.

Stage.	Number of Patients Treated.	Alive with No Evidence of Disease.	Relative Apparent Recovery Rate (%).
Stage I	6,945	4,650	67·0
Stage II	13,386	6,413	47·9
Stage III	12,372	3,359	27·2
Stage IV	2,503	177	7·1
Total	35,206	14,599	41·5

This series relates to material which includes some from the early years of radiotherapy. As was done in Volume 10, it may be of interest to base a study of the results on material from the latest five years as it has been presented in previous Reports.

TABLE V.

Report.	Overall Relative Apparent Recovery Rate for the Last Five Years Embraced by the Report (%).	
	75 Collaborators Reporting in Both Reports.	All Collaborators.
Volume 10	40·4	39·8
Present Report	41·5	41·5

The over-all relative apparent recovery rates in these series are: Volume 6, 36·0%; Volume 7, 36·9%; Volume 8, 38·0%; Volume 9, 38·3%; Volume 10, 39·8%; present Report, 41·5%.

The variation in the six series may be due to (i) the additional number of cases presented by the new collaborators, (ii) differences in the quality of the clinical

TABLE VI.

Comparison of the Five-Year Results Relating to the Last Five Years Embraced by Each Report.

Stage.	Volume 10.		Present Report.	
	Relative Apparent Recovery Rate (%).	Proportion of Cases in the Different Stages (%).	Relative Apparent Recovery Rate (%).	Proportion of Cases in the Different Stages (%).
I	65·4	19·4	66·8	19·9
II	46·8	38·0	47·9	38·0
III	26·4	35·7	27·1	35·1
IV	7·1	6·9	7·1	7·0
Total	40·4	—	41·5	—

material, and finally (iii) to actual improvement in the results obtained.

The following investigation will show the influence of each of these factors. In Table V the apparent recovery rates obtained at the 75 institutions collaborating both in Volume 10 and in the present Report are compared with the corresponding figures for all collaborators.

These figures indicate that the material submitted by the new collaborators, i.e., factor (i), has hardly had any

influence on the total results. Nevertheless, the most reliable appreciation of the importance of factors (ii) and (iii) mentioned above will be obtained by eliminating the new collaborators, thus confining the analysis to the material submitted by the 75 collaborators represented both in Volume 10 and in the present Report, as shown in Table VI.

The cases on which these figures are based number 31,974 in Volume 10 and 34,256 in the present Report. In view of the large number of cases common to both materials, the difference in the over-all relative apparent recovery rates is statistically significant, the "Student" test being on a level of less than 1%.

TABLE VII.
Result of Treatment.

Result.	Total.
Alive with no evidence of the disease	4,642 (53·8%)
Alive with carcinoma present ..	187 (2·2%)
Died of carcinoma	2,996 (34·7%)
Lost sight of	174 (2·0%)
Died from intercurrent disease ..	626 (7·3%)
Total	8,625

Table VI shows that the improvement in the recovery rate from 40·4% to 41·5% is due chiefly to an increased apparent recovery rate in all but one of the stages, and also to a slight improvement in the clinical quality of the material.

TABLE VIII.
Distribution by Stages.

Stage and Group.	Total.
Stage I, Group 1	4,472 (51·9%)
Stage I, Group 2	2,909 (33·7%)
Stage II	1,244 (14·4%)
Total	8,625

If the apparent recovery rates found in this Report are applied to the series of cases in Volume 10, the relative over-all apparent recovery rate in the Volume 10 series would rise from 40·4% to 41·3%.

The difference of 0·9% indicates the average improvement due to increased apparent recovery rates. Using the same method of calculation, it is found that an improvement of 0·2% may be attributed to differences in the quality of the material. Thus, the difference between 40·0% and 41·5% or 1·1% has been satisfactorily accounted for.

TABLE IX.
Relative Five-Year Recovery Rate Calculated for the Various Stages and Groups.

Stage and Group.	Number of Patients Treated.	Alive with No Evidence of the Disease.	Relative Apparent Recovery Rate (%).
Stage I, Group 1	4,472	3,094	69·2
Stage I, Group 2	2,909	1,298	44·6
Stage II	1,244	250	20·1
Total ..	8,625	4,642	53·8

For each of the six last five-year periods the improvement of the results due to an improved clinical quality of the material has been 0·4%, 0·4%, 0·4%, 0·1%, 0·2% and 0·2% respectively. The figures seem to indicate that this improvement, which earlier was constant at 0·4%, has now definitely slackened.

On the other hand, the improvement due to increased apparent recovery rates for the same period has been 0·45%, 0·45%, 0·7%, 0·6%, 1·2% and 0·9% respectively. The slope of this trend must still be considered remarkable.

Carcinoma of the Corpus.

Of the 78 collaborators, 27 have submitted reports on carcinoma of the corpus, including a total of 8625 treated cases.

The five-year results obtained in the treatment of carcinoma of the corpus and the distribution by stages of the cases treated are shown in tabular statements (Tables VII, VIII and IX).

TABLE X.
Carcinoma of the Cervix.

Result of Treatment.	Total.	Latest Five Years.
Alive with no evidence of disease..	334 (36·8%)	136 (44·3%)
Alive with carcinoma present ..	10 (1·1%)	2 (0·6%)
Died of carcinoma	524 (57·8%)	145 (47·3%)
Lost sight of	21 (2·3%)	6 (5·8%)
Died from intercurrent disease ..	18 (2·0%)	18 (2·0%)
Total	907	307

TABLE XI.
Carcinoma of Corpus.

Result of Treatment.	Total.	Latest Five Years.
Alive with no evidence of disease..	126 (59·4%)	66 (59·5%)
Alive with carcinoma present ..	5 (2·4%)	1 (0·9%)
Died of carcinoma	45 (21·2%)	26 (23·4%)
Lost sight of	16 (7·6%)	13 (11·7%)
Died from intercurrent disease ..	20 (9·4%)	5 (4·5%)
Total	212	111

Conclusion.

All figures which have previously been printed in the Report will be available on application to the Editorial Office, Radiumhemmet, Stockholm 60, Sweden, should anybody wish to use them for research work or similar purposes.

Addendum: Figures for Royal Prince Alfred Hospital, Sydney.

The figures for Royal Prince Alfred Hospital are shown in Tables X and XI. These are seen to be slightly above world standard except in so far as the figure for "lost sight of" cases for carcinoma of the corpus was 7·6% against 2% and that for the last five years for carcinoma of the cervix 5·8% against 2·0%. This is partly due to the number of immigrants who are sent to the hospital from the immigration centres and subsequently change their address, and also the language difficulty in answering follow-up letters. The previous figure for "lost sight of" cases for carcinoma of the cervix compiled in December, 1951, was 1%, and in December, 1954, 1·1%.

British Medical Association.

VICTORIAN BRANCH: SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on October 12, 1957, at the Geelong and District Hospital.

The Ubiquitous Staphylococcus.

DR. V. D. PLUECKHAHN read a paper on "The Ubiquitous Staphylococcus" prepared by himself and Dr. Joan Banks (see page 664).

DR. STANLEY WILLIAMS (Melbourne) said that the paper was worthy of any audience anywhere, and the facts produced by Dr. Plueckhahn scared him. Fortunately, however, none of the patients had died, and although staphylococcal infection was widespread, so far it was not terribly dangerous. Perhaps nature, *plus* antibiotics, *plus* proper management would avert morbidity and mortality. Although

reports indicated that some institutions had solved the problem by attention to the nurses' noses, nurses' hands, the visiting doctor, etc., and many publications indicated that preventive measures were successful, Dr. Plueckhahn had shown that they were not. Phage typing gave no indication of sensitivity reaction, and maybe there was a "hospital staph" which did not fit into phage typing. It was possible that staphylococcal pneumonia in children at five to six weeks might well be a hangover from nursery-contracted infections. In the light of the sensitivity tests, he asked what therapy did Dr. Plueckhahn suggest should be used in these cases.

DR. K. C. PORTER (Ballarat) said that Sydney workers had found staphylococci in the alveolar margins as frequently as in the nose, and the question could be asked, "Are we swabbing in the right place?"

DR. B. K. RANK (Melbourne) said that surveys such as Dr. Plueckhahn's were very interesting, but their effect was that antibiotics were to blame, their use should be discontinued, etc. However, it was impossible to deny the value of penicillin cover in, for example, cleft palate operations; and when it was found that that cover was not so good as formerly, penicillin *plus* streptomycin was used, and so on. In other words, "we're on the tiger's back, but let us stay there and do not get a guilt complex because of these surveys".

PROFESSOR MAURICE EWING (Melbourne) said that he had one quarrel with Dr. Plueckhahn's paper: as in Australian meals, there was too much meat in it. As Dr. Plueckhahn had said, staphylococcal infection was not necessarily staphylococcal disease, and it was fortunate that that was so; and it was interesting that the new hospital in Geelong was no better than the old one. He did not agree with Dr. Rank's views. The babies and mothers came in clean, but they were soon infected, and that should not happen. Were the antibiotics to blame? It was in those places where antibiotics were used extensively that there was the most trouble, and perhaps the antibiotics should be locked up for a while.

PROFESSOR HAYDEN asked what was the position of post-operative infection in hospitals. In days gone by, if a hernia wound became infected, the hospital would be turned upside-down, but that was not so now.

Dr. Plueckhahn, in reply to Dr. Williams, said that two, and possibly three, patients had died of staphylococcal infection, one with an infected umbilicus 11 days after replacement transfusion and one at 17 days with fulminating pustular dermatitis; and in the last six months there had been nine cases of osteomyelitis in the children's ward. He was not against the use of antibiotics; but it was the loss of first principles that worried him, and the dependence on antibiotics if things went wrong. Doctors attending the hospital had all been swabbed, and the results were identical with those obtained in the rest of the staff. Personally, he did not think there was a "hospital staph".

In reference to therapy, he thought four antibiotics were useful: penicillin in adequate doses, erythromycin under bacteriological control, chloromycetin, which, despite earlier prejudices, had a real place in therapy, and albomycin, which might be of great use. Post-operative staphylococcal infection was quite common and a very worrying problem, and for a period in individual wards one particular staphylococcus might predominate.

University Intelligence.

AUSTRALIAN NATIONAL UNIVERSITY.

Opening of the John Curtin School of Medical Research.

ON the afternoon of Thursday, March 27, 1958, the permanent building of the John Curtin School of Medical Research at the Australian National University, Canberra, was officially opened by Sir Howard Florey in the presence of the Prime Minister of Australia and other distinguished guests. The Deputy Chairman of the University Council, Dr. H. C. Coombs, presided.

Sir Howard Florey was introduced by the Dean of the John Curtin School, Professor A. H. Ennor, who then invited him to declare the building open.

Before doing so Sir Howard Florey said that Australia had often been called a land of opportunity. Undoubtedly that was true, but opportunity for what? There was plenty of opportunity for sitting in the sun, there were opportunities for making money, opportunities for eating and drinking well—in fact, opportunities of an almost unparalleled kind for the agreeable physical aspects of living. Australia, in the minds of the world in general, was associated with wide open spaces, very large cities, fierce soldiers, horse racing, poor hotels, excellent air services, marvellous tennis players, and at the moment, of course, not quite so marvellous cricketers. But, generally speaking, Australia was not immediately associated with vigorous intellectual activity. That was odd, for Australia produced a high proportion of people of outstanding intellectual attainments. Nevertheless, in comparison with the more advanced countries of the world, Australia was not considered intellectually preeminent, as it was, for example, preeminent in sporting activities. In short Australia had not, so far, been looked upon as a land of opportunity for the intellectual. But things were changing, and on the present day they were celebrating an event which, he was sure, would go down as a landmark in the endeavours of Australia to foster experimental science in a world which was at last beginning to realize the potency of the experimental approach to the study of natural phenomena. The opening of the John Curtin School was a demonstration of the intention of Australia to take its share in the winning of new medical knowledge by experimental means.

Sir Howard Florey went on to say that it was from about the middle of the last century that the great impulse of the application of experiment to medical problems had been first felt. Particularly in Germany and France the foundations had been laid for a physiological, pathological and chemical approach to the phenomena of clinical medicine—in other words to the peculiarities of sick patients. In the present days they were witnessing the invasion of all branches of medicine by the experimental outlook. Few discoveries in medicine were, indeed, now made except by those who utilized experimental methods. However, he did not wish to be misunderstood in the matter. They had with them at the ceremony a very distinguished exponent of clinical investigation, Sir Norman Gregg, who had made, by the use of information collected without experiment in the clinic and examined statistically, a very important observation which was affecting certain aspects of clinical medicine the world over. Exploits as brilliant as his were, however, uncommon. There were, Sir Howard was sure, few who would now dispute that new knowledge was coming principally either from those engaged in what were called the fundamental sciences of medicine or from those who had been trained thoroughly in experimental methods in those sciences and had then gone with the outlook so obtained to work in the clinic. Perhaps he could expand that point a little. He had been so fortunate in his time at Oxford as to have had working in his department some excellent Americans, Australians, Canadians and New Zealanders, mainly Rhodes Scholars. They had pursued various laboratory experimental studies. Many of them when they left had gone back to work in the clinic. Most learnt to think in terms of experiment, and it was gratifying to know that a number of them were now actively adding to knowledge and teaching others as professors of medicine. He hoped that one of the major functions of the John Curtin School would be to train future clinicians in experimental methods and ways of thought as well as to train laboratory workers.

Sir Howard said that he mentioned such matters to put at rest possible lingering fears in Australia that a School of Medical Research could not, by reason of its environment, flourish in Canberra. It entirely depended on what use was made of the present facilities, and it should be kept in mind that research in a hospital in Canberra was not forever barred, but the time for such adventures was not yet ripe.

Turning to the question of how the School had come into being, Sir Howard said that firstly, the finances necessary for its construction had been forthcoming. He thought that the University could count itself extraordinarily fortunate, inasmuch as it was totally dependent on Government money, in finding that all the great political parties of Australia were supporting it. It was perhaps no great secret how much the medical school owed to the constant interest of the present Prime Minister. The school was named after a previous Prime Minister of a different party under whose Government the plan was initiated. All should be greatly encouraged by the fact that the future of the school was not a party matter. The decision had been taken almost 10 years previously to the day to go ahead with the setting up of the school in accordance with the provisions of the Act of Parliament. Preliminary plans were made as to what departments it should house and how they should be manned and constructed. As the Dean had said, the general form of the structure and lay-out of the building was due to Dr. Sanders. The great workshop, which would be the pride of any biological laboratory in the world, was due to him. He took

immense pains to consult people in England and America. For example, Imperial Chemical Industries, who were then planning a great biological laboratory near Manchester, gave freely of their knowledge acquired by extensive investigation. The building also embodied the experience not only of modern new construction but of that gained from Sir Howard's own department which was still, after 30 years, one of the better laboratory buildings in Great Britain. Soon after a decision had been made to proceed with the building of a medical school and the staffing of departments, an attempt was made to find professors for some of the principal chairs which were to be founded. They would thus be available to give their advice not only in the selection of staff but in the construction of their own departments. That plan had, in the main, been followed. At first the professors who were appointed were accommodated in friendly laboratories outside Canberra; one was even housed for more than seven years in London. As there was considerable delay in the construction of the building, temporary laboratories were built in the early 1950's. As it turned out, those had two great advantages; firstly, they brought the scientists who had already been appointed onto the campus in Canberra, and secondly they acted, as it were, as an experiment from which much could be learned about the best internal fittings for the present building. Thus, with the initial plan produced by Dr. Sanders, with the expert knowledge obtained from the professors and from their experience in the temporary laboratories, the architects and engineers for the building were given what few architects were given, fairly precise instructions as to what their scientific clients required. The architects, as all could see, had clothed the ideas of the scientists in an entirely worthy fashion. Great buildings such as the present one did not spring from the ground, especially in Canberra. Many difficulties had had to be overcome. It was not for him to apportion praise or blame in what had gone to the erection of the building. Clearly, the Government had played its part. The Council, Vice-Chancellor and Administration had done all they could to forward the project. The vigour and business acumen of the Dean had been quite invaluable. Sir Howard would like to pay tribute to Mr. Bunker, as he was no longer there. Without him, he thought it was fair to say it would have been extremely difficult to have built the temporary laboratories, certainly to make them as efficient as they turned out to be. Mr. Bunker was a tower of strength especially in the initial stages of the erection of the building. Let him always be remembered with gratitude by those who were fortunate enough to work in it. The architects and builders left a permanent memorial. They would be praised by posterity, for they had made a fine building. It was a building such as he had always dreamed of. It was second to none in the British Commonwealth.

Sir Howard went on to say that the completion and, today, the official opening of the building was, however, only the beginning of the beginning as far as the school was concerned. What was going to be made of the opportunities it afforded? He had peered into his crystal ball, but no certain picture had materialized. The school, as such, had, as yet, little corporate reputation outside Australia. There were two or three outstandingly good Australian scientists there, scientists to whom people came from other parts of the world: scientists who were in the forefront of their subjects. Some young Australians who, in the ordinary course of events, might not have returned from visits abroad, were working there. Thus, a start had been made in carrying out one of the objects of the establishment of the school. It had always to be kept in mind that the school would be judged entirely by the research work that it carried out. Such judgement was not made only locally, it was international and it was not indulgent. The school would have to contend for a long time to come with the pull on young men to go for post-graduate training to the great centres which existed and were rapidly multiplying in the United States and which existed in Europe. If it was to attract and hold the best people, it would need not only to be good but it must be superlatively good, just like the building about to be opened. Sir Howard said that he was a British Commonwealth man himself. He believed in the Commonwealth and that not merely in a flag-waving way. He believed particularly that scientific as well as political bonds should be strengthened between Australia, Great Britain and other Commonwealth countries. The school could play a great part in that process, and he trusted that that aspect of the school's potentiality would be fully exploited, for he felt that without a broad international basis it could soon become parochial. Taking the optimistic view it seemed reasonable to suppose that the school could fail now only from lack of imagination, from selfishness, from a narrow nationalistic outlook, from a failure to integrate itself with the State Universities, and, of course, from lack of cash. Its success would have to be constantly worked for, it would have to be planned for, it would have to be paid for. But how worthwhile it would all be if a growing and vigorous Australia could attract the best Australian scientists to work there, and if a constant traffic of men between different parts of the Commonwealth and foreign countries could be established!

Sir Howard Florey pointed out that the Australian public, who were paying for the institution, would want, understandably enough, to know what was being done with their money. He had indicated briefly some of the prospects, but he urged with all the earnestness that he could command that the public did not look for a series of dazzling and startling discoveries that would have an immediate and profound impact on medicine. It might be years before any such discovery was made—it might never happen. Nevertheless, work of the greatest scientific importance might and almost certainly would go on there: work which the public and Press were not qualified to judge. One of the difficulties of the present time was how to make a literate public understand the mechanism of research that so profoundly affected their lives. It was a problem that had not yet been solved. The public was usually told only of outstanding results or at least of those that were immediately comprehensible or had some dramatic quality. In that connexion he would like to quote a passage from a book by a great microbiologist, Dubos. He was referring to some work of Pasteur on the diseases of silkworms. He said: "The struggle against error, always imminent in these studies on silkworm diseases, is of peculiar interest because it provides a well documented example of the workings of a scientific mind. As Pasteur himself said, it is not without utility to show to the man of the world and to the practical man at what cost the scientist conquers principles, even the simplest and the most modest in appearance. Usually the public sees only the finished result of the scientific effort but remains unaware of the atmosphere of confusion, tentative gropings, frustration and heart-breaking discouragement in which the scientist often labours while trying to extract from the entrails of nature the products and laws which appear so simple and orderly when they finally reach text-books and newspapers."

In conclusion Sir Howard Florey said that he would now declare open the John Curtin School of Medical Research, not with any feelings of overweening pride in the accomplishment of a great building project, but with the hope that they were opening a great intellectual opportunity for future generations of medical scientists.

After the building had been opened, the Prime Minister, the Right Honourable R. G. Menzies, unveiled a commemorative plaque inside the building. Mr. Menzies said that the school commemorated the name of John Curtin, a very great Australian and a very great Prime Minister, a man who at one and the same time was Mr. Menzies's opponent and his friend. Mr. Menzies went on to say that as time went on the school would have a great concentration of mental power. The public should not expect immediate great results from it. Experiment would have to follow experiment, so that ultimately, when the truth emerged, it would be the truth and would have some fundamental quality about it. It was important to appreciate what the school was and what it was intended to do.

Conferring of Degrees.

Earlier in the same day the Australian National University conferred the degree of Doctor of Science (*honoris causa*) on Sir Norman McAlister Gregg and on Sir Howard Walter Florey. The Vice-Chancellor of the University, Sir Leslie Melville, presided.

In presenting Sir Norman Gregg to the Vice-Chancellor, the Dean of the John Curtin School of Medical Research, Profesor E. H. Ennor, said that Sir Norman Gregg was Australia's most distinguished ophthalmic surgeon. He had a high reputation amongst those doctors throughout the world whose especial function it was to attend to the eyes and eyesight. Today, however, they were recognizing Sir Norman for a contribution which went far beyond the confines of ophthalmology—a contribution which had opened up a new and important field in medical science and practice.

Professor Ennor went on to say that in olden times congenital defects in young babies had been ascribed to supernatural influences—the wrath of Heaven or the work of the Devil. Even as late as 1940 the influence of evil spirits had been almost as acceptable an answer as any other to the problem of the origin of such defects. It was Sir Norman's great contribution that he had found, for the first time, a rational explanation for some of those congenital defects; it was his discovery that stimulated a host of other investigations into the physiology and pathology of the

unborn child. In 1941 Sir Norman had been struck by the novel character of a congenital cataract which appeared in Sydney in that year. Similar cases appeared in increasing numbers and over a wide area, and in all of them there were certain similarities. Sir Norman was not satisfied by the then current theories that such defects were due to developmental faults of a genetic character. In his search for an answer to the questions posed by this strange incidence of cataract, he wondered "whether some disease or infection occurring in the mother during pregnancy had interfered with the developing cells of the lens". He then found that the early part of the pregnancies of a number of the mothers whose children were afflicted coincided with an unusually severe epidemic of German measles in 1940. The clinical histories of the mothers involved were then followed up, and Sir Norman found that in all cases the mother had suffered from German measles in the first or second month of pregnancy. At that stage he sought confirmation of what he rightly regarded as a possible solution to the problem. Altogether he collected 78 cases in which both eyes were affected. In 68 of those the mothers had had a rubella infection during the early months of the pregnancy. It was also found that, in addition to the ocular defects, some of the children also suffered from other defects. In 1944 the picture was complete, and such was the accuracy of Sir Norman's observations that a special investigatory committee reported that "there was no evidence of any condition other than rubella as an antecedent of the congenital defects".

In conclusion, Professor Ennor said that the truth of Sir Norman's observations and interpretation had since been abundantly confirmed in all parts of the world and had been recognized by universities at home and abroad and by the Queen when she created him Knight Bachelor. His powers of accurate observation, astute reasoning and dogged perseverance had opened up an entirely new approach to the cause and possible prevention of congenital malformations. It was therefore with great pleasure that he presented to the Vice-Chancellor Norman McAlister Gregg, that he might confer on him the degree of Doctor of Science, *honoris causa*.

The Vice-Chancellor then conferred the degree.

In presenting Sir Howard Florey to the Vice-Chancellor, the Director of the Research School of Physical Sciences, Professor M. L. Oliphant, said that there was no man alive at the present time to whom the nations of the whole world gave greater honour, or to whom all peoples owed a greater debt of gratitude, than Sir Howard Florey. It was his genius which had produced, from the observation of Fleming, the greatest healing agent known to man, penicillin. In common with all men they honoured him for his gift to mankind. They honoured him also for his many contributions to the basic sciences of medicine, and for his achievements as a teacher. But today, they recognized especially his contribution to the University and his devoted efforts in the establishment of the John Curtin School of Medical Research.

In 1944, Sir Howard had come back to his native country as Nuffield Visiting Professor to Australia and New Zealand. As a result of remarks he had made, he was asked to submit proposals for the development of medical research in Australia to Mr. Curtin, the Prime Minister at the time. He made it clear that clinical medicine, like every other branch of applied science, could advance rapidly only by applying fundamental scientific knowledge obtained by painstaking research in the laboratory, and he recommended that a research institute, devoted to the medical sciences, be established in Australia. That report interested greatly a group of imaginative men who saw clearly that the whole future of the country rested upon greater understanding of the basic issues involved. That understanding was to be found in the pursuit of knowledge both in natural science and in the study of humanity and of human institutions. They were able to convince Mr. Chifley, who had succeeded Mr. Curtin as Prime Minister, that a new university should be founded whose primary objective should be research. The Australian National University was established with four research schools, one of which was the institute for medical research recommended by Sir Howard Florey. Sir Howard became not only the father of the John Curtin School of Medical Research, but also one of the founders of the Australian National University. In 1947 Dr. H. C. Coombs, now the Deputy Chairman of the Council of the University, and at that time the most active of its founders, visited Sir Howard in Oxford, and persuaded him to serve with Sir Keith Hancock, Professor Raymond Firth and Professor Oliphant on an Academic Advisory Committee. That committee was formed in Great Britain to advise the interim Council of the academic problems associated with the foundation of the new and unique university. One of its first acts, in which Sir Howard played a leading role, was to press very strongly for the prompt provision of a College and Faculty Club, which would serve to weld the virtually separate research schools into a corporate university, and give a soul to what might otherwise be just another research institution. Sir Howard's far-sightedness helped to give them University House, which would never have been theirs if it had not been the first of the buildings to be erected. During his service on the Academic Advisory Committee, and later as an adviser to the Council, Sir Howard had given abundantly of his time and of his talents to ensure that the staff, the buildings and the equipment of the John Curtin School of Medical Research were of the highest quality. He visited Canberra on several occasions and spared no effort in the task he had set himself. His work on their behalf would bear full fruit in the ceremony in the afternoon, when he would open the laboratories which he designed.

Professor Oliphant said that some years previously the late Sir Edward Mellanby, of the Medical Research Council of Great Britain, had said to him of Sir Howard: "Wherever Florey may be, there good work will be done." That was a singular tribute, to which they in the National University could subscribe without reservation, for they could look at all that he had done for them and know that it was good. They owed him much: they honoured themselves in making him one of them by conferring upon him the degree of Doctor of Science: they would try steadfastly to follow in the course which he had set, towards that goal of understanding which he had followed so fruitfully all his life. Therefore, it gave him the greatest pleasure to present to the Vice-Chancellor Sir Howard Florey, that he might confer on him the degree of Doctor of Science, *honoris causa*.

The Vice-Chancellor then conferred the degree.

Sir Norman Gregg replied on behalf of the Honorary Graduates.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

CREMATION.

[From the *Australasian Medical Gazette*, January, 1893.]

THE first authorized cremation in Australia took place on December 11, 1892, when the body of a Chinese leper was cremated at the Quarantine Ground, Port Nepean. The body was placed in a shell on three tons of wood soaked with kerosene. A light was applied by Dr. Gresswell and in three hours it was burnt almost to a cinder. The few white bones that were left were placed in a small tin box and buried.

Correspondence.

INDICATIONS FOR TONSILLECTOMY.

SIR: Dr. Lopert's article, "Indications for Removal of Tonsils" (April 26, 1958) goes far towards lessening the number of indications for tonsillectomy but surely omits to mention the really important factor which has made review of these indications a current question—the influence of the antibiotics. Dr. Lopert lists five indications for operation all of which in some way mention infection. In nearly every case administration of the appropriate antibiotic (and even now it is likely to be penicillin) would have cured the infection and removed the necessity for operation. Many of the original reasons for operation were derived from the "septic focus" era of medicine, and were the more readily accepted because of the apparent simplicity of the operation and (let's face it) because it is a tempting source of income. The antibiotics have, or should have, put an end to the "septic focus" theory, if it ever had any real basis. In any case, it is no more surgical logic to amputate tonsils because they are subject to recurrent infection than it is to amputate fingers for recurrent whitlows.

Nearly all European migrants have retained their tonsils with little detriment to their health, and it would appear that adults who have them and adults who have not are about

equally healthy. Tonsillar infection and enlargement seem to me to be as much the burden of childhood (and thus of parenthood) as measles, mumps and chicken pox, and for the same reasons. By the age of nine the phase is usually past. Antibiotics should and do reduce the need for tonsillectomy and adenoidectomy to minute proportions, leaving only genuine obstruction to drainage, respiration and swallowing as indications for the removal of organs which, in all probability, have a very useful function.

Yours, etc.,

104 Woodward Street,
Edge Hill,
Cairns,
Queensland.
JOHN MCLEOD.

April 29, 1958.

SUBUNGUAL HÆMATOMA.

SIR: For some ten years I have been using a small right-angle bent cautery heated to barely white heat to make a hole in the nail which has a recent hæmatoma beneath it. Care is taken not to allow the cautery to touch the nail bed, and the cautery is quickly removed after blood appears in the hole so as to avoid overheating the tissues. The whole procedure is then quite painless and very rapid. A sterile result is more likely than with the burr or scalpel technique. If a cautery is not available the head of a darning needle or something similar can be heated in a gas flame to white heat and used with equal effect.

It may well be that this is a widely used method of dealing with this common and very painful condition. However, several recent graduates and the medical superintendent of a metropolitan children's hosptal had not heard of it when I asked them a short time ago, so that publication of this note may help to save some time for casualty residents and general practitioners, and much quite unnecessary suffering for many people.

Yours, etc.,

668 Torrens Road,
Pennington,
South Australia.
ALAN FINGER.

April 16, 1958.

PELVIC EXENTERATION.

SIR: With reference to my article entitled "Carcinoma of the Cervix: The Place of Pelvic Exenteration" (M. J. AUSTRALIA, March 15, 1958) and the letter written by Dr. Philip Corlette (M. J. AUSTRALIA, April 12, 1958), it would appear necessary to draw attention to the following points.

The statement that "Heyman's League of Nations reports of 'Ten Best Centres' show a 25% five-year survival for Stage III and IV cases" is misleading, as results in Stage IV for which the operation of pelvic exenteration was specifically designed, are obscured by including them with Stage III.

The claim that "The Christie Hospital (Manchester, 1945) figures show 29% five-year survival for Stage III and IV cases" is open to question. It is well known that in this part of England, practically all cases of carcinoma of the cervix are referred for treatment by radiotherapy, and yet only 5·9% of this material is classified as being in Stage I, whilst other centres such as Leipzig show 41·2% of cases in Stage I. By incorrect staging, and the inclusion of earlier cases in Stage III and IV, the results in the latter may appear to be better than they actually are. At the Christie Hospital, in cases of carcinoma of the cervix, I have seen radium introduced, without a vaginal examination, by a radiotherapist who freely admits that he has no special knowledge of gynæcology.

The assertion that "there is no selection of cases for radiation therapy" is not substantiated by the authority whom Dr. Corlette quotes, namely Dr. J. Heyman of Stockholm. Figures submitted to him by the Christie Hospital show 8·5% of patients examined but not treated between the years 1933 and 1948, and 7·7% during the next five years. Radiotherapists seldom mention mortality or morbidity resulting from their treatment, but the surgeon is well aware of their prevalence.

It is difficult to compare the results of pelvic exenteration obtained by Dr. Brunschwig at the Memorial Hospital, New York, with cases treated by radiotherapy in Heyman's Annual Reports (now edited by Kottmeier), compiled from 78 institutions in 20 different countries, as a large number of Dr. Brunschwig's cases are cases of failed irradiation where the initial treatment was carried out in other institutions. These patients are specifically excluded from Heyman's Annual Reports. In compiling these reports Heyman admits that diagnosis should be, but in fact is not, based universally on biopsy. As I have already said, "diagnosis by any other means in unacceptable".

To leave the past and return to the present decade, it should be apparent that the clinical staging of carcinoma of the cervix is of limited value. Surgical staging as indicated by Heyman, where the findings at operation are confirmed by histology, including frozen sections taken from the periphery of the indurated area and regional lymph nodes, is of far greater value in assessing the degree of spread of the cancerous process. It is a *sine qua non* that whenever cancer involves a breach of continuity in an epithelial surface, infection always follows and the induration noted clinically may be due to either process, or a combination of both. In the elucidation of this, as Heyman points out, clinical staging is fallacious and surgical staging is the more accurate.

Dr. Corlette agrees with my statement that "radiotherapy is particularly disappointing in the case of recurrent cancer following previous irradiation", and he thinks "it is in such cases, and where radio-resistance has been proven, that pelvic exenteration has most to offer". I would urge, therefore, that these patients be referred for an opinion to someone who is in a position to know and capable of carrying out this type of ultra-radical surgery, rather than be sent away to die.

Yours, etc.,

193 Macquarie Street,
Sydney.
J. CAMERON LOXTON.

April 29, 1958.

NATIONAL SERVICE ACT—INSTRUCTIONS FOR MEDICAL EXAMINATIONS.

SIR: The Department of Labour and National Service is desirous of indicating that a booklet has been issued, February 1958, with instructions for medical examiners carrying out work for that Department, in connexion with the call-up under National Service.

The compilation of the book has been done in association with the Medical Directors General of the Navy, Army and Air Force. The Department of Labour and National Service has requested, therefore, that the existence if this new booklet be brought to the notice of members of the profession, so that they may be aware of its usage in work associated with National Service medical examinations.

Yours, etc.,

L. LOCKWOOD,
Medical Director General (Navy).
W. D. REFSHAUGE,
Director General of Medical Services (Army).
E. A. DALEY,
Director General of Medical Services (R.A.A.F.).

April 28, 1958.

BLOOD WANTED.

SIR: Your leader of March 8, 1958, raises an issue that is of particular importance to country practitioners: when an "immediate" transfusion is required, where is the blood to come from? One suggestion in New South Wales is, I believe, the establishment of regional blood banks at base hospitals. Apart from the risks associated with stored blood, which would naturally multiply with such an organization, one doubts that centralization of this life-saving measure will solve the problem of "immediate" transfusion in towns without base hospitals. Another factor, admittedly not a common one, is the liability of some towns to be isolated by flood. In Singleton we cannot afford to be dependent upon an external supply for this reason alone. But apart from this, we believe that self-sufficiency in a country community is highly desirable. It would be interesting to hear the views of country practitioners elsewhere. As a curtain-raiser, may I describe the operation of the donor panel in this town.

When donors enrol, they are venesected by local doctors, and the blood samples (five cubic centimetres) forwarded for testing to the Red Cross blood transfusion service, who provide the necessary collecting tubes. Apart from typing and serological tests, this ensures that any donor who leaves the town may enrol with a similar organization anywhere else without inconvenience. A membership card is forwarded to each donor by the transfusion service, and a donor card bearing his relevant particulars is received and filed by the

local donor secretary, who for the past ten years has been the superintendent of Singleton district ambulance. Various people may act in this capacity, but in our opinion he is the man for the job, for reasons that will presently unfold. All donor cards are in his keeping, and he sees to it that his officers are thoroughly familiar with them. Doctors also should be familiar with the blood-grouping nomenclature used by the Red Cross, and with the corresponding colours of the donor cards. If a group A donor is wanted, for instance, the ambulance staff will be confused by a request for group A(2), or worse still, for group 2.

When a transfusion is required, the doctor telephones the ambulance officer on duty and specifies the number, and perhaps the sex, of donors required. The blood group is specified by asking for "A for apple: blue card", or "O for orange: white card", and so on. The task of going through the cards, collecting donors, and delivering them one by one to hospital with their cards (and to their homes later) is done most efficiently by the ambulance staff, while the doctor can watch the patient and set up for compatibility testing and venesection. This system works so well that the first donor often arrives before we are ready for him. Each donor is venesected on a bed, and not in the operating theatre.

Although routine blood-grouping is done for us by a pathology service at Maitland, thirty miles away, here again self-sufficiency is necessary for "immediate" blood-grouping and cross-matching, including the Rh factor. Nor is it sufficient that only one person in the town should be able to do this. The polyvidone technique has solved a great problem for general practitioners in this regard, and although one could not desire anything more explicit than the description given by Walsh and Ward,[1] one of whom originated the method, it should be learned at first hand from actual demonstration, and by practice under supervision.

The further problem of pyrogenic reactions is virtually eliminated by having apparatus prepared by one or two nurses who are specially trained in the procedure laid down by the same authors.[2] It should be eliminated altogether by using a "prefabricated" plastic set such as the "Fenwal Blood Pack".

The "reluctance to come forward for blood donation" to which, Sir, you have referred is not apparent in this town. We have three hundred donors, and from time to time the numbers are replenished and increased by a "donor drive", in which we receive strong support from the local Press. Perhaps there is a moral in this that the medical profession should remember: many people who are reluctant to serve as donors for a central "pool" are more than willing to serve their own community when the need arises.

Yours, etc.,
P. W. GILL.

Dangar Road,
Singleton,
New South Wales.
April 29, 1958.

[1] "A Guide to Blood Transfusion", by R. J. Walsh and H. K. Ward, 1957. Sydney: Australian Red Cross Society. Pages 107 and 120.

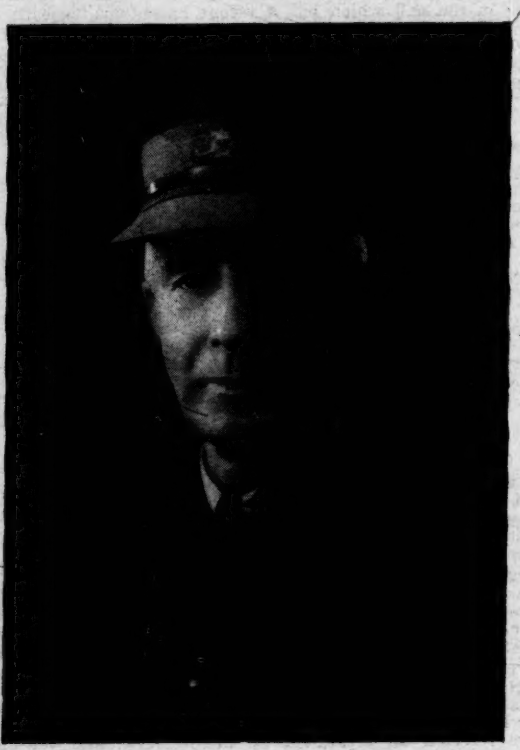
[2] *Ibidem*, appendix 2, pages 153 to 156.

Obituary.

ALLAN SEYMOUR WALKER.

ALLAN SEYMOUR WALKER was born on June 19, 1887, at Richmond, New South Wales. Both his father and his mother had served in the Education Department of New South Wales and his father, Thomas Walker, later became the Senior Inspector of Schools for the State. Allan Walker received his school education in the New South Wales State schools. He gained a high school scholarship in 1901, passed the junior university examination in 1903, with seven A's and the medal in English, and the senior university examination in 1905, with seven A's and three B's, being *proxime accessit* for the General Proficiency Prize and receiving the medal in English and honours in Latin, French and mathematics. He entered the Medical School of the University of Sydney in 1906, obtained honours at the annual examinations each year and graduated M.B., Ch.M., in 1910 with first class honours. He was then for two years on the resident medical staff of the Royal Prince Alfred Hospital, being successively a junior and a senior resident medical officer. In 1913 he entered into a period of private practice at Parkes, New South Wales, which lasted until 1920. During that time he was occupied in home service in the Australian Army Medical Corps during 1917. In 1921 he began work in the Department of Pathology of the University of Sydney Medical School under the Commonwealth Department of Health, and in 1922 the University conferred on him a Doctorate in Medicine in Pathology, with first class honours and the University Medal. A further period of private practice at Summer Hill in the suburbs of Sydney lasted from 1922 to 1927, apart from a visit to England for post-graduate study in 1926. In 1927 he commenced practice as a consulting physician in Sydney, and this continued until 1945, although he was on war service from 1939 to 1945. He held a series of honorary hospital and teaching appointments. At the Royal Prince Alfred Hospital he was Honorary Morbid Anatomist from 1922 to 1923, Honorary Assistant Physician from 1923 to 1937, Honorary Physician from 1937 to 1945, and Honorary Consulting Physician from 1945 till his death. He was also Honorary Consulting Physician of the Western Suburbs, St. George, Canterbury District and Parramatta District Hospitals. He was a part-time demonstrator in pathology in the University of Sydney from 1923 to 1937 and a lecturer in clinical medicine from 1937 to 1945.

His association with the Australian Army Medical Corps began in 1917, when he was appointed as a Captain and he held an Honorary Captaincy from 1919 to 1937, when he



became an Honorary Major. In 1939 he joined the A.I.F. and was appointed a Lieutenant-Colonel. He served in the Middle East as Senior Physician with the 2/1 Australian General Hospital from 1939 to 1941, and on his return to Australia was appointed Senior Physician at the Concord Military Hospital in 1942. In the same year he was appointed Consulting Physician to the Australian Army with the rank of Colonel, which he held until 1944. From that year until the time of his death he was an Honorary Colonel in the Royal Australian Army Medical Corps. His military service took him not only to the Middle East, but to New Guinea and other areas, and he was twice mentioned in dispatches.

Allan Walker was also active in the service of his profession. He was a member of the Council of the New South Wales Branch of the British Medical Association from 1934 to 1939. He took an active part in many Australasian Medical Congresses, being an office-bearer of various sections on a number of occasions. His most notable activity, however, was amongst his fellow physicians. In 1930 "representative" physicians of Australia had combined with some of the leading physicians of New Zealand to form a body known as the "Association of Physicians of Australasia including New Zealand"—a curious description which

bore witness to the reluctance of New Zealand physicians to be dubbed "Australasian". Allan Walker was too junior then to be a leading spirit in this formation, but he was one of the first group invited to join this body. He was its honorary secretary in 1938 when the Association became merged into The Royal Australasian College of Physicians with headquarters located in Sydney, and it was a logical step when he was elected to the honorary secretaryship of the new College. Only those who worked with him in the formative days of the College know the splendid work which Walker put into its organization. The inaugural meeting which took place in the Great Hall of the University of Sydney in a scene of surpassing pageantry and the series of night and day celebrations which followed, all done on a grand scale, were in large measure due to his painstaking activity and guidance. Further, he set a standard of executive work in the College which succeeding honorary secretaries were pleased to follow. On his enlistment in the Australian Imperial Force early in 1940 he was granted leave by the College in the hope that he would return to office after a short period of service abroad, but he remained in the Army till the war was over, and he never resumed his executive duties. He continued, however, to serve on the Council till 1956, when owing to illness he decided not to contest another Council election. Meanwhile in the years 1946-1948 he had the honour of holding the position of a Vice-President of the College. From 1949 to 1952 he was the representative of the College on the National Health and Medical Research Council.

The last, but perhaps the most notable phase of Allan Walker's career began in 1945 when he was appointed editor of the Medical War History of the Australian Army in the War of 1939 to 1945. He brought to it great gifts of knowledge, experience and skill; his discriminating mind, his training in medicine and in the Army and his unusually fluent pen equipped him in a remarkable way for the difficult and arduous task. He worked steadily, despite considerable practical difficulties and often indifferent health, and maintained an interest not only in his own immediate task, but in the broader matter of the preparation of medical war histories throughout the world. He was the Australian representative from 1946 until his death on the Liaison Committee of Medical Historians of the British Commonwealth. The three volumes that have been published, "Clinical Problems of War", "Middle East and Far East" and "The Island Campaigns", stand as a memorial to him. The fourth volume, dealing with the medical services of the Royal Australian Navy and the Royal Australian Air Force, has not yet been published, but he had managed to do a good deal of the ground work for it before his death, which occurred on January 8, 1958, after a long illness.

This long and impressive but somewhat bald account of Allan Walker's many offices and achievements gives little impression of the man himself and of the charm of his personality. These will be better seen in the tributes which follow. It is sufficient to say that those who knew him well esteemed him highly as a man and as a friend. The gifts that he brought to his work as medical war historian were for many years at the service of this journal, to which he contributed a great many articles, particularly in the editorial columns. His loss created a great gap in the circles of those who knew him, but he has left a legacy which will ensure that he is not forgotten. Our sincere sympathy is extended to his widow and family.

The following verses, in his own handwriting, were found among Allan Walker's papers:

"Hooray" exclaims the white-haired boy,
Viewing the past decades,
"Let's count up every shiny toy
I've had ere come the shades.

"I think another game I'll start
That I can each morn play,
Some ritual not requiring art
Practised at break of day.

"I've been a cockerel that struts
Upon the stage of life,
Now unkind fate my feathers cuts
With time's insistent knife.

"So let me play, 'twill do no harm
If every day at dawn
I rise and sound my mild alarm
Across the dewy lawn.

"I'll flap my shortened useless wings
As once I used to do,
While from my wisened throat there flings
A cock-a-doodle doo."

PROFESSOR LORIMER DODS writes: My first memories of Allan Walker belong to the year 1921 when I met him as one of my demonstrators in pathology in the old medical school—a rather shy and slender young man in a spotless white coat, who was very helpful and understanding as a teacher, and who spoke of the art and practice of internal medicine with a fervour and enthusiasm which left an indelible impression on at least one member of his student audience. About 15 years later, in 1936, I had the pleasure of attending his ward rounds at the Royal Prince Alfred Hospital for several months and learned something of his surprisingly wide knowledge of bed-side and laboratory medicine and was most impressed by his practical and thoughtful interest in the social, economic and emotional problems of each individual under his care—to quote his own words: "It is so easy to generalize on diagnosis or treatment but so difficult to appraise the special needs of one sick man." Many of us who worked in close association with Allan Walker in the 2/1 Australian General Hospital during the first two years of the last war have very happy memories of his kind and helpful guidance, and of his warm and friendly loyalty to his medical juniors. During these years I had the privilege of talking to him about many things and learned to appreciate his sensitive and cultured mind, his love of music, his breadth of thought and reading, and his natural kindliness. On more than one occasion he told me that he would not want to return to the private practice of medicine after the war and that he would like to devote his remaining years to some form of medical journalism. Fortunately the opportunity for this type of work came very soon after the war, and I have a vivid memory of the surprisingly youthful and happy excitement with which he told me of his appointment as Australian medical historian. I also know something of the deep satisfaction which he found in the writing of the three volumes of Australian medical history which now stand as his special memorial.

MR. GAVIN LONG writes: In Allan Walker Australia was fortunate to find a medical war historian who was not only a medical scientist whose learning was both wide and deep but a man of rare literary ability, widely read, imaginative, and master of a fastidious and fluent literary style. The temperaments both of the scientist and of the artist found expression in his life and in his work. Some indication of the sort of reading that had helped him to shape his style and his philosophy is given in the quotations woven into the reflective passages that occur at intervals throughout his work: from Amiel, Chesterton, Lamb, Arnold Bennett, Auden, T. S. Eliot, Constant Lambert, as well as Burton and Pasteur.

It may seem an odd remark to make about a history of medical services in a war, but from Allan Walker's volumes could be compiled a valuable small book for apprentice historians, giving fine examples of narrative writing, analytical writing, descriptive writing, reflective writing and so on. As a narrator his sensitive and imaginative touch is seen at its best in such passages as the one about the embarkation from Crete; or the one headed "Over the Mountain Passes" in the account of the operations in Greece; or the one about action round Kokoda. In his reflective passages his judgements are gentle yet firm, and studded with incisive phrases: "the Norns of modern medicine", for example; "that dubious entity the 'herd instinct'"; "a service national in outlook and individual in application"; "humanity with its warm spiritual metabolism"; "the comfortable shrine of mechanolatry". Here was no mere chronicler, though his description of incidents and organization was precise and lucid. Here was a philosophical scientist-historian, with something very definite to say and the gift of saying it so that the layman as well as the doctor would understand and remember.

It is hard writing that makes easy reading. Allan Walker wrote and rewrote, revised and revised, somewhat in the manner G. M. Trevelyan describes in his autobiography. And he worked always at high pressure. In the early days we had hoped that much of the clinical volume at least might be contributed by others, but this was not to be, though, as he explained, he was greatly helped by many colleagues who provided notes and criticism. It was, naturally, a great encouragement to him that his books were so warmly received both in Australia and overseas, and that the demand for them was far greater than we had expected. Both "Clinical Problems of War" and "Middle East and Far East" had to be reprinted within two or three years of publication.

DR. LAURENCE HUGHES writes: I would like to contribute a personal appreciation of the late Dr. Allan Walker as I have had the privilege of a sincere friendship with him and his family for many years and a great admiration for his character and intellectual ability. Allan and I were

not fellow-undergraduates in medicine, but in the early nineteen-twenties we became associated in post-graduate studies and as honorary physicians at the Royal Prince Alfred Hospital. He was a man of culture with a wide knowledge of medical and classical literature and more than considerable interest in music and the arts, and his contributions to medical literature were numerous. When these rich endowments were supplemented by his experiences on active service in the 1940's it was natural that he should have been chosen to write the medical history of Australia in the war of 1939-1945. Although this task, together with the after-effects of illness contracted on service in the Middle East, no doubt contributed largely to his final illness, the work was undertaken and completed in thorough and characteristic fashion and will remain an all-time memorial to him. *Exegi monumentum ære perennius.* This distinction not withstanding, his family and friends, patients, and colleagues like to cherish the memory of Allan Walker for the respect and affection in which he was held, his loyalty, honesty, and "nameless unremembered acts of kindness" which form the "best part of a good man's life".

VIVIAN MORRIS RICH.

WE are indebted to Dr. J. C. Storey for the following account of the career of the late Dr. Vivian Morris Rich.

In the early hours of Thursday, April 17, 1958, Sydney lost one of her most faithful and sincere general practitioners. Vivian Rich died peacefully in his sleep about 6.30 in the morning. Educated at Sydney Grammar School, he had as contemporary students Roy Sear and the writer. He matriculated in 1905 and proceeded to the University of Sydney Medical School. He qualified in 1909 and sailed to England where he was resident medical officer at Prince of Wales Hospital, Tottenham, and at the Children's Hospital, Chadwell. At the start of the first World War he enlisted in the Royal Army Medical Corps and served for a time at Malta. He then went to France as a regimental medical officer and ambulance officer. He was promoted to the rank of Major and decorated with the *Croix-de-Guerre* and star for bravery in the field. He was wounded in action once. He went with the army of occupation to Cologne, returning to Sydney in December 1919.

In 1920 he commenced practice at Edgecliff, and in 1922 at 530 Old South Head Road, Bellevue Hill, where he enjoyed a very busy practice till 1946, when he retired owing to ill-health. Having a keen sense of duty he responded to every call and at times was greatly overworked. All his patients had loved him and were greatly upset at his retirement.

A keen golfer, he won the B.M.A. cup in 1935, but in 1946, on medical advice he changed to bowls, joining the Rose Bay Club. He also played at the New South Wales Club, La Perouse, where he often acted as host to many of us. Rich introduced to the Eastern Suburbs Medical Society a fund to increase the incomes of the wives of men away at the war.

Having known Vivian for over sixty years I feel qualified to speak of his fine character. Never have I known him to lose his temper and he was tolerant and thoughtful of everybody. He earned the love and gratitude of all his patients. He leaves a widow and three children to whom we extend our sincere sympathy.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes etc. are published in the *Commonwealth of Australia Gazette*, No. 26, of May 1, 1958.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps (Medical).—1/8079 Captain (provisionally) H. A. Muir is appointed from the Citizen Military Forces, Royal Australian Army Medical Corps (Medical), Northern Command, and to be Captain, 10th March, 1958, with a Short Service Commission for a period of one year. To be Major, 13th February, 1958, with a Short Service Commission for a period of two years.—4/8048 Harry Adair Kreiser. To be Captains, 7th February, 1958, with Short Service Commissions for a period of two years and three months.—3/12030 Samuel Henry Richard Palmer, 3/12032 Charles Norman Matthews, and 3/12031 John Richard Thomson. To be Captain, 3rd May, 1958.—2/40168 Lieutenant (Temporary Captain) W. F. Hammond.

Regular Army Special Reserve.

Royal Australian Army Medical Corps (Medical).— The appointment of SX700106 Lieutenant V. P. Danius is terminated, 31st December, 1957.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—1/39197 Captain (provisionally) N. F. Langley relinquishes the provisional rank of Captain, 10th March, 1958, is transferred to the Reserve of Officers (Royal Australian Army Medical Corp (Medical) (Northern Command), in the honorary rank of Captain, 11th March, 1958. 1/39192 Captain (provisionally) H. A. Muir is appointed to the Australian Regular Army, 10th March, 1958. The following officers are appointed from the Reserve of Officers, and to be Captains (provisionally):—Honorary Captains 1/59814 B. E. Todd, 4th March, 1958, and 1/39215 R. V. Graham, 7th March, 1958. 1/39205 Captain (provisionally) J. P. Waller relinquishes the provisional rank of Captain, 11th February, 1958, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Northern Command) in the honorary rank of Captain, 12th February, 1958. To be Major, 13th March, 1958.—1/46764 Captain A. J. Kerr.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—2/50436 Captain J. J. G. McGirr is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command), 1st March, 1957. F2/2804 Captain (provisionally) J. Hughes relinquishes the provisional rank of Captain, 20th February, 1958, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command), and is granted the honorary rank of Captain, 21st February, 1958. To be Colonel, 1st March, 1958—2/206960 Lieutenant-Colonel H. O. G. Selle. To be Captain (provisionally), 4th March, 1958—2/127060 Daryl Frederick Booth. The provisional ranks of the following officers are confirmed:—Captains 2/79254 R. J. M. Atkinson and 2/79316 F. H. Lang. 2/79316 Captain F. H. Lang is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command), 12th March, 1958. To be Captain (provisionally), 19th March, 1958—2/206962 Roderick Donald Macdonald.

Southern Command.

Royal Australian Army Medical Corps (Medical).—The appointment of 3/101017 Major B. H. Gandevia is terminated, 1st December, 1957. To be Captain (provisionally), 4th March, 1958—F3/1036 Dorothy Barbara Newton. The provisional ranks of the following officers are confirmed:—Captains 3/123506 D. A. Jolley, 3/101035 G. J. Little, and 3/101036 H. D. Breidahl.

Western Command.

Royal Australian Army Medical Corps (Medical).— 5/26529 Captain (provisionally) M. Traub is seconded whilst in the United Kingdom, 30th March, 1958. The provisional appointments of the following officers are terminated:—Captains (provisionally) 5/26552 R. F. Gorman, 23rd February, 1958. and 5/26529 M. Traub, 29th March, 1958. To be Captains (provisionally)—5/26552 Richard Francis Gorman, 24th February, 1958, and 5/26529 Max Traub, 30th March, 1958.

Tasmania Command.

Royal Australian Army Medical Corps (Medical).—6/15393 Captain (provisionally) P. Pitney relinquishes the provisional rank of Captain, 2nd February, 1958, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Tasmania Command), and is granted the honorary rank of Captain, 3rd February, 1958. 6/15392 Captain (provisionally) L. M. Jacks relinquishes the provisional rank of Captain, 18th February, 1958, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Tasmania Command) in the honorary rank of Captain, 19th February, 1958.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

Northern Command.—To be Honorary Captain, 7th March, 1958—John Hardwick Phillips.

The following officers are placed upon the Retired List with permission to retain their rank and wear the prescribed uniform, 30th June, 1958:—

Northern Command.—Lieutenant G. L. Alison.

Southern Command.—Majors T. S. Gregory and C. L. Rosefield, Captain R. M. Trewhella, E.D., and Lieutenant H. C. B. Henshall.

Western Command.—Lieutenant-Colonel A. F. Janes.

The following officer is placed upon the Retired List (Tasmania Command) with permission to retain his rank and wear the prescribed uniform, 1st May, 1958: Major J. L. Grove, C.B.E.

The following officers are retired:—

Northern Command.—Honorary Captain E. J. Ryan, 30th June, 1958.

Southern Command.—Honorary Captains J. Clough and C. B. Melville, 30th June, 1958.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Annual Subscription Course.

DR. PAUL DUDLEY WHITE, Physician to the Massachusetts General Hospital, Boston, President of the International Society of Cardiology, Director of the United States Public Health Institutes, and Physician to President Eisenhower, will visit Australia under the auspices of the Cardiac Society of Australia and New Zealand. In conjunction with the Post-Graduate Committee in Medicine, Dr. White will give the following lecture: "Heart Disease the World Over", on Monday, May 26, at 8.15 p.m. in the Wallace Theatre, University of Sydney.

Course in Anæsthesia for General Practitioners.

The Post-Graduate Committee in Medicine in the University of Sydney announces that a Course in Anæsthesia for general practitioners will be conducted by the Department of Anæsthetics and Resuscitation at the Sydney Hospital for two weeks from November 17 to 28, 1958. The Course will be a full-time one from 9 a.m. to 6 p.m. daily, consisting of practical demonstrations and tutorials. Enrolments will be limited to three and applicants should submit particulars of their anæsthetic experience and requirements. The closing date for applications is July 31, 1958, and the selection of candidates will be announced shortly after this date. Fee for attendance is £12 12s., payable after selection date. Written application, enclosing full particulars, should be sent to the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 5535, BU 4497-8. Telegraphic Address: "Postgrad Sydney".

Post-Graduate Conference at Bathurst.

The Post-Graduate Committee in Medicine in the University of Sydney, in conjunction with the Western District Medical Association, will hold a post-graduate conference at the Bathurst Town Hall on Saturday and Sunday, June 7 and 8, 1958. The programme is as follows:

Saturday, June 7: 2 p.m., registration; 2.30 p.m., "Virus diseases of the central nervous system", Dr. W. J. Burke; 4 p.m., "Some surgical aspects of peripheral vascular disease", Dr. G. D. Tracy.

Sunday, June 8: 9.30 a.m., "Recent advances in therapeutics", Dr. A. W. Morrow; 11 a.m., "Modern trends in the management of cerebral vascular disease", Dr. W. J. Burke; 12 noon, "Common diseases of the upper abdomen and their management', Dr. A. W. Morrow.

Fee for attendance at the Course is £3 3s., and those wishing to attend are requested to notify Dr. Brooke Moore, 142 William Street, Bathurst, as soon as possible. Telephone: Bathurst 2345.

Post-Graduate Conference at Tamworth.

The Post-Graduate Committee in Medicine in the University of Sydney, in conjunction with the Northern District

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED APRIL 26, 1958.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	1	5	..	..	..	..	..	7
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	4	..	4
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	5	..	1	..	..	..	..	..	6
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	10(1)	8(7)	1(1)	1	1	..	1	1	23
Diphtheria	..	..	..	..	1	..	..	..	1
Dysentery (Bacillary)	..	26(1)	3(1)	..	2(2)	..	4	..	35
Encephalitis	..	..	..	1	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	2	..	..	2
Infective Hepatitis	30(10)	16(5)	10(2)	1(1)	14(1)	1	..	..	72
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	2	..	..	..	2
Leptospirosis	2	..	5(1)	..	..	..	..	..	7
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	1(1)	1	..	..	..	1	..	..	3
Ophthalmia	..	..	..	..	3	..	..	..	3
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	..	..	..	..	..	..	..	..
Puerperal Fever	1	..	..	..	..	..	..	..	1
Rubella	..	15(12)	..	1	5(4)	..	..	..	21
Salmonella Infection	..	..	..	1(1)	..	..	..	..	1
Scarlet Fever	17(5)	35(26)	6(1)	1(1)	3(2)	1	..	3	66
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1(1)	..	..	..	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	29(12)	14(11)	10(6)	7(4)	5(5)	4(3)	2	..	71
Typhoid Fever	1(1)	..	..	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Medical Association, will hold a week-end conference at the Tamworth Base Hospital on Saturday and Sunday, June 7 and 8, 1958. The programme is as follows:

Saturday, June 7: 9 a.m., at the Tamworth Golf Club, Maguire Cup (Ladies) and Poidevin Cup (Gentlemen); 2 p.m., at the Tamworth Base Hospital, registration; 2.30 p.m., "Some common diagnostic problems in skin disease", Dr. W. Keith Myers; 4 p.m., "Allergy: Diagnosis", Dr. S. E. L. Stening.

Sunday, June 8: 9.30 a.m., "Sterility: Investigation or operation?", Dr. Robert Mackey; 11 a.m., "Practical points in dermatological treatment", Dr. W. Keith Myers; 12 noon, "Allergy: Treatment and management", Dr. S. E. L. Stening; 2.30 p.m., "Minor disabilities of pregnancy", Dr. Robert Mackey.

Fee for attendance at the Course is £3 3s., and those wishing to attend are requested to notify Dr. G. B. Dance, President, Northern District Medical Association, 454 Peel Street, Tamworth, 4N, as soon as possible. Telephone: F 735.

Medical Prizes.

THE DARCY COWAN PRIZE.

THE Darcy Cowan Prize has been founded by the Australian Laennec Society (the medical section of the National Association for the Prevention of Tuberculosis in Australia) to honour the work of Sir Darcy Cowan, a prominent Adelaide physician in the field of tuberculosis in Australia. It is expected that this prize will be offered each year and that it will be awarded first at the next national meeting of N.A.P.T.A. in Melbourne in February, 1959.

The particulars of this prize are as follows: (i) Its value is £50. (ii) It is to be awarded for the best essay on any subject relating to respiratory disease. It should include original work and observations. (iii) It is open to all medical graduates of Australian universities of not more than five years' standing or thirty years of age.

Essays (three copies of double-spaced typing on foolscap) must be in the hands of the National Secretary by November 30, 1958. They should be addressed to Dr. R. Munro Ford, 163 North Terrace, Adelaide, South Australia. Further particulars concerning this prize may be obtained from Dr. Ford.

The College of Radiologists of Australasia.

EXAMINATION FOR DIPLOMA.

THE College of Radiologists of Australasia will hold examinations for Part I and Part II of the Diploma of the College, commencing on August 25, 1958. The examination for Part I will be held in the candidate's own State, and that for Part II will be held in Sydney. Full details and application forms are available from the office of the College, British Medical Agency, Twelfth Floor, 135 Macquarie Street, Sydney. Entry forms must be received at the office of the College in Sydney not later than June 30, 1958.

Medical Appointments.

The following appointments have been made to the honorary medical staff of the Royal Alexandra Hospital for Children, Sydney: Honorary Assistant Physician, Dr. A. R. Tink; Honorary Relieving Assistant Physicians, Dr. J. D. Harley and Dr. C. A. Rigg; Honorary Urologist, Dr. M. S. S. Earlam; Honorary Assistant Urologist, Dr. R. D. Smith; Honorary Assistant Ear, Nose and Throat Surgeon, Dr. O. J. Davies.

Deaths.

THE following death has been announced:

CORKHILL.—Arthur Basil Corkhill, on April 30, 1958, at Kilsyth, Victoria.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Cutler, Geoffrey Abbott, M.B., B.S., 1952 (Univ. Sydney), 10 Addison Road, Manly, New South Wales.

Baltcailis, Margers Bertolds, M.D., 1935 (Univ. Riga), regional registration for Ivanhoe region, Post Office, Ivanhoe, New South Wales.

Diary for the Month.

MAY 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 21.—Western Australian Branch, B.M.A.: General Meeting.
MAY 22.—Victorian Branch, B.M.A.: Executive Meeting.
MAY 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
MAY 23.—Queensland Branch, B.M.A.: Council Meeting.
MAY 27.—New South Wales Branch, B.M.A.: Hospitals Committee.
MAY 28.—Victorian Branch, B.M.A.: Council Meeting.
MAY 29.—South Australian Branch, B.M.A.: Clinical Meeting.
MAY 29.—New South Wales Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales. Anti-Tuberculosis Association of New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

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